CPC Emergency Medicine

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Clinical Practice and Cases in Emergency Medicine

In Collaboration with the *Western Journal of Emergency Medicine*

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In Collaboration with the *Western Journal of Emergency Medicine*

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Clinical Practice and Cases in Emergency Medicine

In Collaboration with the *Western Journal of Emergency Medicine*

JOURNAL FOCUS

Clinical Practice and Cases in Emergency Medicine (CPC-EM) is an internationally recognized journal affiliated with the MEDLINE-indexed *Western Journal of Emergency Medicine* (*West*JEM). It will offer the latest in patient care case reports, images in the field of emergency medicine and state of the art clinicopathological cases. CPC-EM is fully open-access, peer reviewed, well indexed and available anywhere with an internet connection. CPC-EM encourages submissions from junior authors, established faculty, and residents of established and developing emergency medicine programs throughout the world.

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Policies for peer review, author instructions, conflicts of interest and human and animal subjects protections can be found online at www.cpcem.org.

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VITAL STATISTICS

Clinical Practice and Cases in Emergency Medicine (CPC-EM) is an internationally recognized journal affiliated with the MEDLINE-indexed *Western Journal of Emergency Medicine (West*JEM). CPC-EM is distributed electronically to 23,278 emergency medicine scholars. This includes our sponsors: California ACEP, the American College of Osteopathic Emergency Physicians, California Chapter of AAEM, and over 80 academic department of emergency medicine subscribers as well as six AAEM State Chapters.

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Announcing New Case Report and Image journal: *Clinical Practice and Cases in Emergency Medicine* (CPC-EM)

Mark I. Langdorf, MD, MHPE University of California, Irvine Medical Center, Department of Emergency Medicine, Orange, California

We are pleased to announce that the Editorial Board of the *Western Journal of Emergency Medicine* has decided to begin a new segment journal affiliated with *West*JEM but focused on case reports and images. The new open access journal will be titled: "*CPC-EM*" which stands for *Clinical Practice and Cases in Emergency Medicine*.

Dr. Rick McPheeters, Chief of Emergency Medicine at Kern Medical Center, and Vice Chair of Emergency Medicine at UCLA, will serve as Editor in Chief of the new journal, while Dr. Shadi Lahham, Associate Program Director from UC Irvine will serve as Associate Editor.

*West*JEM is spinning off the case reports and images from the core journal for three important reasons.

First, we want to continue to encourage submissions from junior authors, both faculty and residents. Most residents or medical students contemplating an academic career write a case report as their first scholarly project. We want to continue to provide an outlet for these reports, as this is one of the core missions of the journal, to mentor scholarship from new and developing EM programs and faculty. There are precious few outlets for case reports and images in our specialty, and many journals charge substantial fees for publication. *West*JEM has seen the number of case reports and image submissions increase dramatically over the past few years, and given constraints of space we have, sadly, had to decline more than 80% of these recently. There is clearly a demand out there.

Secondly, as *West*JEM pursues an official "impact factor," publication of case reports and images work against us. A journal's impact factor is a ratio of the number of citations of papers in a journal divided by the total number of papers published. Case reports and images are rarely if ever cited, and yet contribute to the denominator of the impact factor calculation. The more case reports and images, the lower the impact factor. So it makes sense to spin off these rarely-if-ever cited communications so as not to dilute the core journal's impact factor. Although *West*JEM does not yet have an official impact factor from the international core index from Thomson Reuters, our self-calculated impact factor is 1.208 (1.208 citations in *West*JEM and other journals in 2014 and 2015 per article published in *West*JEM in 2013).

And lastly, case reports, for which there is clearly a large demand, can support the core journal and its activities to provide the best scholarship to the world. As an open-access journal, *West*JEM relies on society sponsors (ACOEP, California ACEP, Cal-AAEM and UC Irvine Department of EM) and 73 department sponsors, along with modest article processing fees (APF) of $400 per paper. Recognizing that case reports are important, and also use journal staff time, we have decided to maintain the APF of $400 per case report or image, and offer a 15% discount to department sponsors. In a perfect world, we would waive the APF for case reports and images as in the past for department sponsors. In the fiscal reality of 2016, we need to use case reports to support finances for the core journal.

The new journal, *CPC-EM*, will be included in PubMed Central, and also in PubMed abstract search services, just as is the core *West*JEM.

We are excited about *CPC-EM* and believe it will both continue to contribute to junior scholarship, as well as provide resources to grow *West*JEM to a bigger and better journal.

Best regards,

Mark I. Langdorf, MD, MHPE
Editor in Chief, *West*JEM

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Conflicts of Interest: By the *CPC-EM* article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

Clinical Practice and Cases in Emergency Medicine: Birth of a New Journal

Rick A. McPheeters, DO Kern Medical, Department of Emergency Medicine, Bakersfield, CA

I'm extremely excited to be part of this new segment journal affiliated with *Western Journal of Emergency Medicine:* Integrating Emergency Care with Population Health (*West*JEM). The advent of this new journal will only have positive effects which will be many-fold. Primarily, however, it will invite the best medical science to *West*JEM and yet still give a much needed and larger outlet for lower tiered clinical evidence of case reports and images. Although known to be at the bottom of the hierarchy, they certainly serve a purpose in the medical literature.

Historically, the recognized great scholarly professions included medicine, law and the clergy. As many of us in academic medicine have heard the common phrase of "Publish or Perish," we have realized there is some truth to this statement. In years past at the University of Notre Dame, priests serving as instructors were expected to produce scholarly works. If they did not, they were re-assigned as parish priests outside of the university. As the phrase was subsequently embraced by non-sectarian entities, the "a" was replaced by an "e". Academic medicine is quite similar where "Perish" means lack of faculty appointment and/or promotion, or in the setting of a residency training program, a citation from the Residency Review Committee of Emergency Medicine.

The current acceptance rate for case reports and image submissions with *West*JEM, ranges between ten and twenty percent, reflecting that upwards of 90% of these manuscripts have been declined and not necessarily because they were of poor quality or unimportant but predominantly because of limited print space as well as their dilution of *West*JEM's impact factor. This new journal will allow for a greater acceptance rate, therefore helping us fulfill one of our main missions of advancing academic emergency medicine by recognizing the scholarly work most associated with resident physicians and junior faculty.

Secondarily, and in time, this move should increase the impact factor of the parent journal, and as such, the volume and quality of submitted research articles should greatly enhance the open-access dissemination of important scholarly work worldwide. These are certainly exiting times and with that said, I would like to introduce and welcome you to the inaugural on-line edition of *Clinical Practice and Cases in Emergency Medicine*, also known as CPC-EM!

Respectfully,

Rick A. McPheeters, DO
Editor in Chief, *CPC-EM*

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Conflicts of Interest: By the *CPC-EM* article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

18-year-old Female with a Change in Mental Status

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CASE PRESENTATION

An 18-year-old female presented to the emergency department (ED) with confusion and "abnormal behavior." Her family stated that she'd had increasingly abnormal speech for one week, word-finding difficulties, and then required frequent redirection to complete tasks. Two weeks prior to presentation, the patient was involved in a motor vehicle crash (MVC). The vehicles were moving slowly and sustained little damage. The airbags did not deploy and the patient was belted. She subsequently developed a series of headaches that persisted for a week and then resolved. Two days before presenting to our ED, she was seen at another ED for intermittent, crampy abdominal pain accompanied by vomiting, fevers to 38.9°C, and headache. She was diagnosed with a urinary tract infection (UTI) and discharged, but she did not take the nitrofurantoin she was prescribed. These symptoms continued on the day of presentation to our ED.

She had a past medical history of sickle cell trait. She did not take any medications and she had no drug allergies. Her family history was notable for epilepsy and systemic lupus erythematosus. She denied any tobacco, alcohol, or illicit drug use. She was a recent high school graduate.

She was alert and in no acute distress on physical exam. She was afebrile (37.1°C) with a heart rate of 90 beats/minute, blood pressure 135/85 mmHg, and her oxygen saturation was 99% while breathing room air. She weighed 100.5 kg and was five feet, six inches in height, had a body mass index of 36.9 kg/m^2, and was well developed and well nourished. Her head was normocephalic and atraumatic, and mucus membranes were dry. Pupils were equal, round and reactive to light and accommodation; the extra ocular movements were normal. Sclera were anicteric and fundi were without papilledema. The neck was supple and without lymphadenopathy or carotid bruits. Her lungs had coarse breath sounds bilaterally but no wheezes, crackles, or rhonchi. There were no retractions or increased work of breathing. Heart was regular, rate and rhythm, without murmurs, rubs or gallops. The abdomen was soft with normal bowel sounds and without distention, tenderness, rebound or guarding. There was no costovertebral angle tenderness. The extremities had no edema, had 2+ pulses and were without tenderness or deformity.

Neurologic examination showed cranial nerves II-XII intact, 5/5 strength throughout all extremities, normal muscle bulk/tone and intact sensation. Her speech was clear. The patient was noted to be withdrawn and have a flat affect. She followed commands, answered most questions appropriately but occasionally was confused. She was oriented to self but not to place or time.

Initial laboratory results are shown in Tables 1-3. Because of her mental status changes and history of fever, a lumbar puncture was done. Her opening pressure was 12 cmH_20. The results are shown in Table 4. A diagnostic test was then performed, which confirmed the diagnosis.

Table 1. Complete blood cell count of 18-year-old patient presenting with mental status changes, fever, and a family history of autoimmune disease.

Complete blood cell count	
White blood cells	6.6 K/mcL
Hemoglobin	12.4 g/dL
Hematocrit	36.8%
Platelets	251 K/mcL
Differential	
Polymorphonuclear leukocytes	52%
Lymphocytes	44%
Monocytes	3%
Eosinophils	1%

Table 2. Chemistry results.

Serum chemistries	
Sodium	141 mmol/L
Potassium	3.3 mmol/L
Chloride	104 mmol/L
Bicarbonate	27 mmol/L
Blood urea nitrogen	7 mg/dL
Magnesium	0.65mg/dL
Phosphorous	2.5 mg/dL
Total protein	7.1g/dL
Albumin	3.8 g/dL
Total bilirubin	0.7 mg/dL
Aspartate aminotransferase (AST)	26 u/L
Alanine aminotransferase (ALT)	25 u/L
Alkaline phosphatase	66 units/L
Additional Labs	
Thyroid stimulating hormone (TSH)	1.73 mIU/L
C-reactive protein (CRP)	0.34 mg/L
Erythrocyte sedimentation rate (ESR)	8 mm/hour
Coagulation Studies	
Prothrombin time (PT)	15.9 seconds
Partial thromboblastin time (INR)	31 seconds
International normalized ratio	1.2

Table 3. Urinalysis results.

Urinalysis	
pH	6.2
Specific gravity	1.020
Glucose	Negative
Ketones	Negative
Nitrites	2+
Leukocyte esterase	Negative
White blood cells	Trace
Red blood cells	0-5 count/uL

Table 4. Cerebrospinal fluid results.

Cerebrospinal fluid	
Glucose	75 mg/dL
Protein	29 mg/dL
White blood cells	12K count/uL
Red blood cells	3K count/uL
Polymorphonuclear leukocytes	17%
Lymphocytes	78%
Monocytes	5%
Gram stain	Negative
Opening pressure	12 cm H_2O

CASE DISCUSSION

When I first read this case, I realized that there was a lot of information to sort through. Specifically, in the history of present illness (HPI), there were several keywords that drew my attention:

18 year-old female presenting with confusion and "abnormal behavior" - abnormal speech, word-finding difficulties, and need for frequent redirection of tasks, for approximately one week.

She also had intermittent crampy abdominal pain, vomiting, intermittent fevers to 102°F, and headache for three days.

She was seen two days prior for similar complaints and diagnosed with a UTI, but did not fill her prescription.

Fourteen days prior she was involved in a minor MVC and subsequently developed headaches that persisted for a week and then resolved.

The physical exam was most remarkable for just a few findings: she had dry mucous membranes and a flat, withdrawn affect; she had ability to follow commands and answer most questions appropriately; but occasionally she was confused and oriented to self but not to place or time.

In an attempt to analyze this information, I went back to my mnemonic for mental status change, "I WATCH DEATH."

Infectious
Withdrawal
Acute metabolic disorder
Trauma
Central nervous system (CNS) pathology
Hypoxia
Deficiencies
Endocrinopathies
Acute vascular
Toxins, substance use, medication
Heavy metals

Infectious etiology: Here the differential includes CNS infections (meningitis, encephalitis) and any systemic infection. The patient had a history of fever but was afebrile in the ED, and her white blood cell count (WBC), C-reactive protein and sedimentation rate were all normal. Her urinalysis was inconsistent with an infectious source. The cerebrospinal fluid (CSF), however, did have white blood cells present, but only 12, which is atypical of an acute infection. So, for now, I will keep infection on my list of possibilities but continue to look elsewhere for answers.

Withdrawal: Alcohol and benzodiazepine withdrawal can cause mental status changes. Given the patient's lack of

substance abuse history, coupled with a duration of symptoms greater than one week with gradual worsening, these causes are significantly less likely.

Acute metabolic disorders: Disturbances in sodium, calcium, or magnesium levels, as well as hepatic and renal failure, can present with altered mental status. The normal serum electrolyte levels, and normal renal and liver functions, excluded this possibility.

Trauma: I know that the patient had a minor MVC two weeks prior to presentation, so the differential must include post-concussive syndrome, as well as a possible subdural, epidural, or subarachnoid hemorrhage. Dr. Crouter described a minor MVC, which would have a low likelihood of causing an intracranial hemorrhage in an 18-year-old adult. Furthermore, the patient was now headache-free and did not have a decreased level of consciousness. Post-concussive syndrome was a possibility, but that became a diagnosis of exclusion given the other, more dangerous possible causes of the patient's complaints.

CNS pathology: Non-traumatic etiologies in this category include stroke, hemorrhage, tumor and seizure. Stroke, hemorrhage and a space-occupying lesion are all unlikely, as you would expect to see focality in the neurologic examination. The patient's family seemed attentive to her and no seizure activity was reported. Although focal seizures and a post-ictal state were possible, the duration of symptoms made this highly unlikely.

Hypoxia: This one is easy. The patient had a documented pulse oximetry (POx) of 99%. Although carbon monoxide poisoning can cause a normal POx reading in the setting of hypoxia, the patient had progressive symptoms in a variety of different settings. This is therefore removed as a possibility.

Vitamin **D**eficiencies: This category includes B12 and thiamine deficiencies. The patient was described as well developed and nourished, and there was no reported history of substance abuse. Although levels would need to be obtained to fully eliminate these as possibilities, these diagnoses are extremely unlikely. This category is therefore off of my list.

Endocrinopathies: Here I had to consider disorders of the thyroid, parathyroid and adrenal glands, as well as diabetes mellitus. The thyroid stimulating hormone and calcium levels were within normal limits, eliminating thyroid and parathyroid disease. The sodium, potassium and glucose were also normal, eliminating both diabetes and an adrenal crisis.

Acute vascular disorders: Options in this category include shock, hypertensive encephalopathy and acute myocardial infarction. The patient's complaints and hemodynamics do not support any of these etiologies. This category is off of my list.

Toxins, substance use, and medications: This category is broad and must be carefully considered. Although substances such as alcohol, anticholinergics and synthetic marijuana must be considered, the duration of her symptoms made all of these unlikely. For the patient's symptoms to be ongoing and progressive over one week would require repeated exposures to these substances, including after she became confused. Her parents reported that she required redirection to complete tasks, so self-administration of substance is highly unlikely. This category is off my list.

Heavy metals: The patient did not work in industry or a laboratory. There is no reason, from the history, to think she had a heavy metal exposure.

I have eliminated all categories except for "infectious." Now it was time for me to get out my magnifying glass and look at my clues a bit more closely. I started by re-examining the CSF results. The CSF had a WBC count of 12 cells/microL. With bacterial or viral meningitis, you anticipate counts of at least 100 cells/microL. Lower cell counts can, however, be seen with early bacterial meningitis, viral meningitis, neurosyphilis, tuberculous meningitis and encephalitis. The duration of symptoms, coupled with a negative gram stain, eliminated early bacterial meningitis. Given the normal glucose and protein on CSF analysis, tuberculous meningitis was excluded. Non-congenital neurosyphilis is extremely rare in this age group and a normal CSF protein essentially excludes this diagnosis. The normal protein also makes a viral etiology less likely. The patient's profound mental status changes were much more suggestive of encephalitis, so I will continue to hunt for clues that support or refute this diagnosis.

We know the patient had just graduated from high school, so it is a reasonable assumption that her vaccinations were up to date. This minimizes the risk of her having encephalitis from measles, mumps, or rubella. It is important to remember that the patient also complained of abdominal pain and was recently diagnosed with a UTI. Could that "UTI" really have been a herpes simplex virus (HSV) outbreak? Considering this, HSV encephalitis is high on my list.

Of the non-infectious (aseptic) encephalitis etiologies, the one of greatest concern here is autoimmune. I am told there is a family history of autoimmune disease (lupus), which puts this patient at higher risk.

The two remaining possibilities are HSV encephalitis and autoimmune encephalitis. The abdominal pain, nausea and vomiting have yet to be explained and this, I believe, holds the key to the final diagnosis. Autoimmune disease can be triggered by viral infections, such as gastritis.

In review, this was an 18-year-old female with signs and symptoms consistent with encephalitis and a family history of autoimmune disease, who recently had a gastrointestinal illness. Bringing this all together, my final diagnosis is autoimmune encephalitis triggered by a systemic viral infection of the gastrointestinal tract. The diagnostic test is a magnetic resonance image (MRI) of the brain.

CASE OUTCOME

The diagnostic study was an MRI of the brain. As described by the radiologist, the patient had patchy foci of T2 FLAIR hyperintensity and diffusion restriction, primarily involving the medial temporal lobes bilaterally, with additional smaller scattered areas throughout both cerebral hemispheres. Diagnostic considerations include encephalitis such as infectious or limbic/paraneoplastic (patient images shown in Images 1 and 2). Empiric vancomycin, ceftriaxone, and acyclovir were given and the patient was admitted to the intensive care unit (ICU). Her mental status continued to deteriorate, requiring mechanical ventilation as she began to have multiple seizures. The next day she began treatment for suspected autoimmune encephalitis with intravenous immunoglobulin (IVIG) methylprednisolone, and plasmapheresis. She began having refractory seizures on hospital day (HD) three, ultimately requiring levetiracetam, divalproex sodium, phenytoin, and pentobarbital drips. A tracheostomy tube was placed. On HD 18, a CSF autoantibody panel revealed autoantibodies to glutamic acid decarboxylase (GAD). The team planned to start rituximab therapy for GAD autoimmune encephalitis, but this was delayed by persistent fevers. On HD 29 she received her first dose of rituximab therapy, and on subsequent days she was able to be weaned from her antiepileptic regimen and gradually recovered neurologic function. On HD 34, the patient was weaned off of the ventilator. She was speaking through her Passy-Muir tracheostomy valve, texting, and laughing with family. She was transferred to rehab after her second dose of rituximab on HD 36. She was discharged home after her fourth and final dose of rituximab on HD 50. Per her family, she regained her cognitive baseline. Her tracheostomy was successfully reversed two weeks later.

RESIDENT DISCUSSION

Encephalitis is an acute inflammation of the brain, and approximately 10% of the 20,000 cases seen in the United States annually are fatal.[1-3] The primary injury occurs as the brain parenchyma suffers a widespread inflammatory response, causing edema, hemorrhage, and the destruction of neurons.

Data is limited in encephalitis as cases are rare and the testing required to make the diagnosis is expensive, slow, and inconsistently performed. Fifty percent of cases have no identifiable pathogen.[1,2] When an etiology is identified, viruses are the most common cause.[4] These viruses include HSV (10-14% of all cases worldwide), eastern and western equine encephalitis virus, West Nile virus, non-polio enteroviruses, varicella-zoster virus and rabies.[2-5] Autoimmune-related etiologies are becoming recognized as an important cause of encephalitis and may account for up to one-third of cases.[6] Autoimmune encephalitis can result from patients developing antibodies to voltage-gated potassium channels, N-methyl-D-aspartate (NMDA) receptors, gamma-aminobutyric acid (GABA) receptors, or glutamic acid decarboxylase (GAD) receptors.[5,7]

Autoimmune encephalitis tends to affect the limbic system (amygdala, hippocampus, hypothalamus), causing emotional lability, personality changes, and decreased memory.[8] Patients may even have delusional or paranoid thoughts, visual or auditory hallucinations and seizures.[2] It often progresses insidiously over a period of days to weeks, with patients eventually presenting to medical providers with seizures or headaches.[2] Viral infections can set off a variety of inflammatory and autoimmune disorders such as systemic lupus erythematosus, Sjogren's syndrome, Hashimoto thyroiditis and autoimmune encephalopathy.

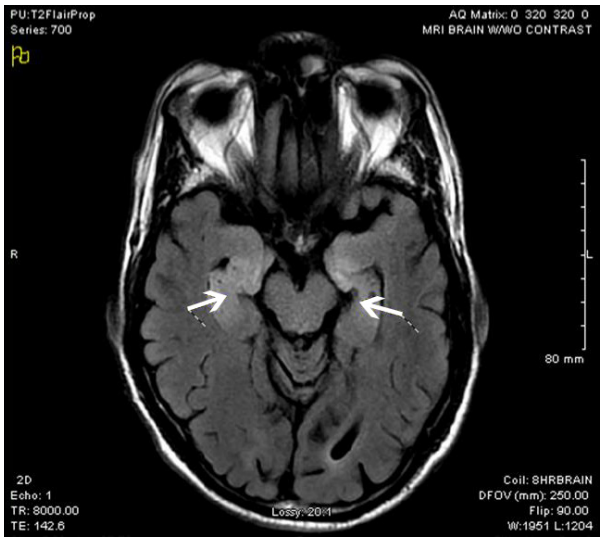

Image 1. Axial T2 FLAIR sequence magnetic resonance imaging showing bilateral enhancement of the hippocampus (arrows). *FLAIR,* fluid attenuated inversion recovery

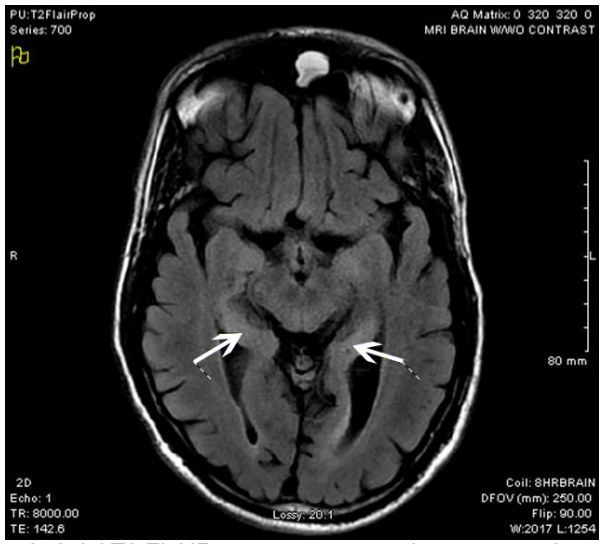

Image 2. Axial T2 FLAIR sequence magnetic resonance imaging showing enhancement of the medial temporal lobe (arrows). *FLAIR,* fluid attenuated inversion recovery

The ED workup includes a set of basic labs, which often reveals a nonspecific elevation in the WBC count. Ammonia, carboxyhemoglobin level, toxicological screen, and blood cultures should all be measured to rule out alternative etiologies for the patient's presentation. Studies of the CSF are often normal, regardless of the underlying etiology of the encephalitis. The most common abnormalities seen on CSF analysis are lymphocytic pleocytosis, an elevated protein level and oligoclonal bands.[8,9] Computed tomography of the head is the first imaging study to order, as it will rule out other etiologies of altered mental status, such as space-occupying lesions and intracranial hemorrhage. MRI is the diagnostic imaging modality of choice as it is the most sensitive for encephalitis.[9,10] T2-weighted and FLAIR sequences may show hyperintense signal and mild swelling of the medial temporal lobes,[8] as it did in our patient.

Patients with encephalitis should therefore be admitted to the hospital for further evaluation, diagnostic studies, and management. Initial management in the ED includes supporting the patient's airway, breathing, and circulation. Empiric antibiotics and acyclovir should be administered, since HSV encephalitis can have similar MRI findings.[8] Seizures are common and are managed initially with benzodiazepines. Refractory seizures and status epilepticus can be treated with second- and third-line agents such as phenytoin, fosphenytoin, and phenobarbital. Inflammation and vasogenic edema caused by the encephalitis can lead to increased intracranial pressure that can be life threatening.[2] Patients may require mannitol or hypertonic saline administration when there is a concern for elevated intracranial pressure, and the initial evaluation should include a measurement of the patient's opening pressure on lumbar puncture. Patients should undergo additional CSF studies as part of their inpatient workup to differentiate between the various causes of encephalitis. Many CSF autoantibody studies may take days to weeks to result, and empiric treatment for autoimmune and infectious etiologies is appropriate preceding or in the absence of a confirmed diagnosis.

First-line treatment for autoimmune encephalitis consists of the administration of steroids (ex. methylprednisolone 30mg/kg/day) plus IVIG (0.4 g/kg/day) or plasmapheresis.[11] Second-line treatment options include immunomodulators such as rituximab and cyclophosphamide.[11,12,13] Patients with suspected autoimmune encephalitis should undergo a workup for underlying malignancy as a part of their admission.[14] Both NMDA and GAD autoimmune encephalitis are associated with ovarian teratomas and gastrointestinal malignancies, respectively.

Prognosis for encephalitis is extremely variable based on the specific underlying etiology. Mortality for viral encephalitis ranges from <1% in cases of non-polio enteroviral encephalitis, to approximately 50% for eastern equine encephalitis, to nearly 100% for rabies encephalitis.[2] Autoimmune encephalitis has a relatively good prognosis with proper treatment. There is a 10% mortality, and 81% of patients have favorable outcomes.[2]

FINAL DIAGNOSES

Autoimmune limbic encephalitis due to glutamic acid decarboxylase antibodies, complicated by delirium and status epilepticus.

TAKE-HOME POINTS

- Encephalitis is a life-threatening disease with many potential causes.
- History and physical examination are critical to making the diagnosis.
- Autoimmune encephalitis
    - Presents insidiously.
        - Personality changes, psychiatric symptoms, headaches, and seizures are the most common presenting complaints.
    - Lymphocytosis may be seen on CSF testing.
        - Neither sensitive nor specific.
    - CT imaging of the brain is a reasonable initial choice to rule out competing diagnoses.
    - MRI is the diagnostic imaging study of choice.
        - T2-weighted and FLAIR sequence images may show hyperintense signal and mild swelling of the medial temporal lobes.

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Sleep-Associated Torsades de Pointes: A Case Report

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Torsades de Pointes (TdP) is a polymorphic ventricular tachycardia that occurs in the presence of an acquired or congenital long QT syndrome (LQTS). We present the case of a 57 year-old man with end-stage renal disease on methadone maintenance in which there occurred multiple episodes of TdP during sleep. The patient was found to have a QTc interval of 548 milliseconds, and the dysrhythmia was successfully treated with isoproterenol infusion and methadone substitution. It is surmised that the patient had a multifactorial, acquired LQTS that during somnolence, reached a critical threshold of QT prolongation to lead to the development of TdP. [Clin Pract Cases Emerg Med. 2017;1(1):9-12.]

INTRODUCTION

Sudden cardiac death (SCD) is a major cause of death worldwide, with a reported incidence in the U.S. of more than 400,000 cases per year. One uncommon cause of SCD is torsades de pointes (TdP), defined as polymorphic ventricular tachycardia occurring in a patient with an acquired or congenital long QT syndrome (LQTS).[1] TdP accounts for fewer than 5% of SCD cases, and death presumably occurs from its degeneration into ventricular fibrillation.[1] Medications with QT-prolonging effects are the most frequent cause of acquired LQTS, which is estimated to occur with approximately 2-3% of all prescriptions written.[2,3] Another cause of acquired LQTS involves electrolyte disturbances, including hypokalemia, hypocalcemia, and hypomagnesemia.[4,5] Furthermore, LQTS may be a result of cardiac abnormalities such as bradycardia with atrioventricular (AV) block, congestive heart failure, ischemic heart disease, rheumatic heart disease, myocarditis, and mitral valve prolapse.[4,5] This case report discusses a patient in a normal physiologic state of sleep that may have contributed to the development of TdP associated with an acquired LQTS.

CASE REPORT

A 57 year-old man with past medical history of hypertension, diabetes mellitus, dyslipidemia, and end-stage renal disease was transferred from his dialysis center after suffering a cardiac arrest. According to the center's staff, he experienced ventricular fibrillation for which he was immediately defibrillated with an automated external defibrillator. During the chest compressions that followed, the patient promptly regained consciousness. After the successful resuscitation, he was transferred to the emergency department (ED) by emergency medical services.

Upon arrival to the ED, the patient had no complaints. He recounted that during his dialysis session, he "was watching TV, got tired and fell asleep, and was woken up by the shock." He reported no prodromal symptoms prior to the event. His maintenance medications included simvastatin, clonidine, insulin, and methadone. The patient had an elevated blood pressure of 160/84, heart rate of 85, respiratory rate of 20, and temperature of 97.6 °F. His oxygen saturation was 97% on nasal cannula at three liters of oxygen per minute. Mental status was normal, and skin was normally perfused. Cardiorespiratory examination was normal except for the presence of a dialysis catheter in the right side of the chest. Pupils were 1-2 millimeters bilaterally.

Aspirin 325 mg was given orally. The initial electrocardiogram (ECG) revealed sinus rhythm with junctional bigeminy, an occasional premature ventricular complex, left ventricular enlargement, and a significantly prolonged QTc of 548 milliseconds (Image 1). Continuous cardiac telemetry was initiated, and electrophysiology consultation was sought with the plan of hospital admission to the coronary care unit (CCU). Serum potassium was measured at 3.6 mEq/L and magnesium was 2.0 mg/dL, both of which were within the normal ranges.

Approximately three hours into the ED course, the patient lost consciousness, and the monitor demonstrated ventricular fibrillation (VF). Immediate defibrillation at 200J was performed, and the patient quickly regained consciousness

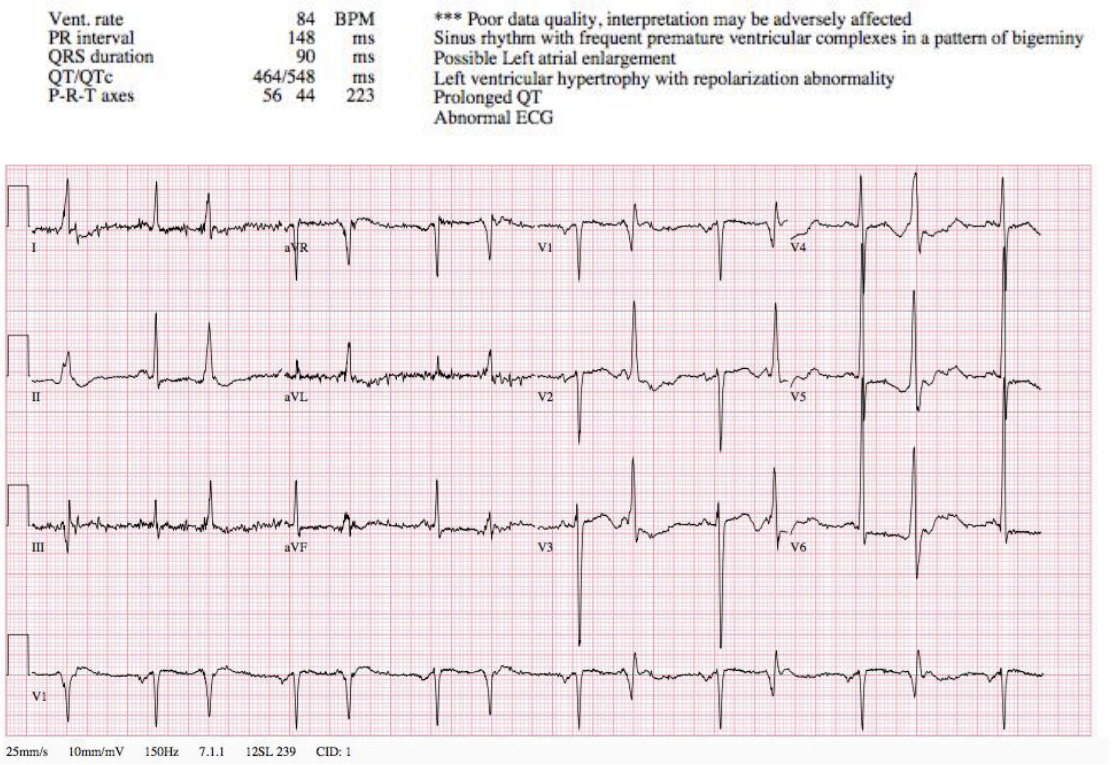

Image 1. Initial ECG in the emergency department showing prolonged QTc of 548 msec.
ECG, electrocardiogram

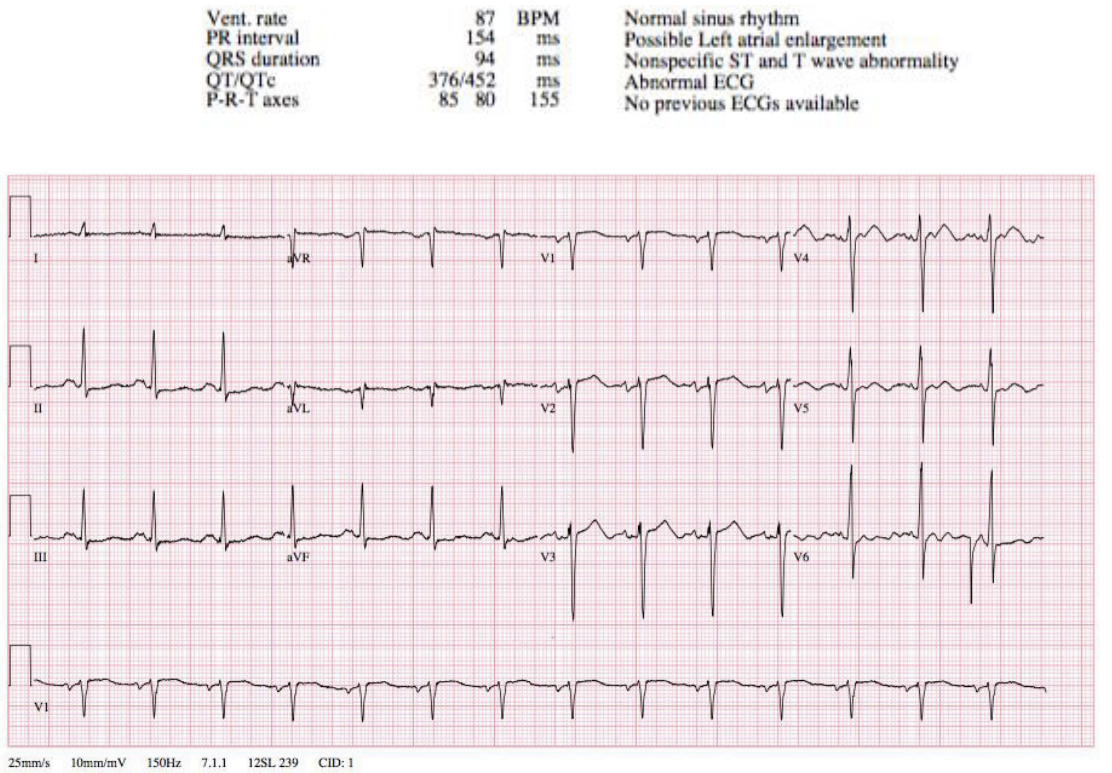

Image 2. Repeat ECG in the coronary care unit after methadone cessation showing normal QTc of 452 msec.
ECG, electrocardiogram

during subsequent chest compressions. A repeat examination was unchanged. Due to presumed recurrent VF, metoprolol 5 mg was given intravenously to treat possible electrical storm. Minutes later, the physician at the bedside observed that the patient had become somnolent. The monitor showed sinus bradycardia, which was succeeded by polymorphic ventricular tachycardia. The physician again successfully defibrillated the patient. The diagnosis of TdP was now presumed, and a magnesium infusion of 1 gram was administered over 30 minutes. The patient experienced one more identical arrest sequence, and upon recommendation by cardiology, a lidocaine infusion was started. The patient was transferred to the CCU.

During the CCU course, methadone dose was slowly reduced. In spite of the lidocaine infusion, the patient continued to have occasional bradycardia episodes during somnolence. As a result, the lidocaine infusion was discontinued, and an isoproterenol infusion was initiated. The patient's QTc remained prolonged on the lower methadone dose, so the drug was discontinued and substituted with buprenorphine/naloxone. The patient had no more ventricular fibrillation episodes, and a repeat ECG revealed a QTc of 450 milliseconds (Image 2). Serial troponins and electrolytes continued to be normal, and an echocardiogram indicated systolic dysfunction with an ejection fraction of 30-35%. Additional hospital records included the results of a cardiac catheterization three weeks prior that showed non-obstructed coronary arteries. The isoproterenol infusion was gradually tapered, and the patient was fitted for a wearable defibrillator. He was discharged on hospital day 12 with continued outpatient treatment for opioid dependence and plan for possible automated implantable cardioverter-defibrillator placement.

DISCUSSION

Pathophysiology

The QT interval represents a portion of the cardiac action potential, mainly ventricular repolarization.[2,6] If a factor such as a medication or electrolyte abnormality was to delay repolarization, this would manifest as a prolonged QT interval on the ECG. During such prolongation of the repolarization phase, there is a destabilization in membrane channels that may give rise to an afterdepolarization.[7,8] An afterdepolarization is an oscillation of the membrane potential of one cell that occurs as a result of the upstroke of an action potential of a nearby, stimulated cell.[7,8] If an afterdepolarization is strong enough to bring the cell membrane to its threshold potential, it will result in a depolarization. This depolarization, when it occurs during repolarization, is commonly known as the "R on T phenomenon," the inciting event for the TdP dysrhythmia.[8] Furthermore, since this rhythm is a response to a preceding impulse, it is referred to as a triggered activity.[8]

Back to Our Case

This patient had numerous risk factors for TdP. He presented with a significantly prolonged QTc and then experienced episodes of sinus bradycardia. The patient had both a prolonged QT and QTc, suggesting that other factors, beyond the heart rate, were contributors to delayed repolarization. Methadone is one of many medications that have been shown to directly influence cardiac repolarization,[6,9,10] and even low doses of methadone (<100mg) have been shown to cause QT prolongation.[6,9,10]

There is an inverse relationship between heart rate and duration of repolarization.[2] Slow heart rates, even benign, sinus bradycardia, are characterized by longer repolarization times and therefore longer QT intervals.[2] QT prolongation may also result from drug-induced bradycardia or pathological causes such as high-grade AV blocks or sick sinus syndrome.[2,11] In this case, TdP was observed when the patient fell asleep, and somnolence itself is characterized by bradycardia.[12,13,14] Polysomnography of healthy individuals has detected bradycardia to as low as 30 beats per minute and sinus pauses averaging from 2-11 seconds, both of which occur during early non-REM and REM sleep stages.[12,13,14] This is hypothesized to be a result of either decreased adrenergic tone or increased vagal stimulation that occurs during physiologic sleep.[12,13] Holter electrocardiography of healthy subjects has also shown that *both* the QT *and* the QTc intervals increase during somnolence, along with episodes of bradycardia and sinus pauses during sleep.[15] It is therefore suggested that heart rate changes may not be the only contributor to the QT prolongation during sleep, and that sleep itself may be an independent, QT-prolonging factor.[15] While we were not able to obtain a rhythm strip during each episode that captured the exact onset of TdP, one of the authors directly observed the dysrhythmic event to be remarkably coincident with somnolence. It is therefore reasonable to suspect that the recurrent dysrhythmia occurred due to a further prolonged QT as a result of sleep, directly and/or through sleep-associated bradycardia or sinus pause.

Lastly, the procedure of hemodialysis, interestingly, can lead to QT changes, aside from the electrolyte abnormalities alone.[16] Prolongation of the QT and decreased adaptability of QT interval to variations in heart rate have been reported to occur during dialysis sessions.[16] Sudden cardiac death may occur, and if so, occurs most often in the immediate period after a hemodialysis session during which altered ventricular repolarization can persist for several hours.[16] Although our patient's electrolytes were found to be within normal limits at time of presentation, the lingering cardiac effects of his recent hemodialysis session may have played a role in the development of TdP.

CONCLUSION

This case describes an interesting presentation of TdP, presumably due to a variety of factors that resulted in an acquired long QT syndrome. There are numerous risk factors for the acquired form of LQTS, with novel causes being reported each year.[17,18,19] However, what distinguished this

case were the recurrences of TdP during somnolence. While previous studies have suggested a theoretical mechanism for TdP during sleep, we are not aware of any cases in the literature.[1,7,13] This patient ultimately did well with judicious treatment including prompt electrical therapy, magnesium, isoproterenol, and methadone cessation.

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Weber B Distal Fibular Fracture Diagnosed by Point-of-care Ultrasound

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We report the case of a 45-year-old woman who presented to the emergency department (ED) after an acute ankle inversion injury. After history and physical exam suggested a potential fracture, point- of-care ultrasound (POCUS) demonstrated a cortical defect of the distal fibula, consistent with fracture. Plain radiography failed to demonstrate a fracture. Later, the fracture was identified as a Weber B distal fibular fracture by stress-view radiography. This case reviews the evaluation of acute ankle injuries in the ED and the utility of POCUS as a supplemental imaging modality in the evaluation of ankle fracture. [Clin Pract Cases Emerg Med. 2017;1(1):13–15.]

INTRODUCTION

Ankle injuries are a common presenting complaint in the emergency department (ED), comprising approximately 5% of all ED visits.[1] It is important to accurately differentiate ankle fractures and other serious injuries requiring orthopedic consultation from less serious injuries that may be managed conservatively. The current standard of care for the evaluation and diagnosis of ankle fracture, when indicated by the Ottawa Ankle Rules (OARs), is plain film radiography. The OARs are designed to decrease radiographic evaluation of ankle injuries; they have a high sensitivity but very low specificity for fracture diagnosis. This results in many false positives with increased radiation exposure and additional costs. Additionally, plain film radiography is not completely sensitive for ankle fracture, resulting in false negatives. Furthermore, radiographic imaging necessitates radiation exposure and additional cost. This report describes the value of point-of-care ultrasound (POCUS) as a supplemental imaging modality in the evaluation of ankle injuries to aid in making the diagnosis of ankle fracture.

CASE REPORT

A 45-year-old previously healthy woman presented to the ED with a chief complaint of left ankle pain after a fall while roller skating, which resulted in an inversion injury to the left ankle. The patient presented approximately one hour after the injury. She reported no history of surgery or trauma to the ankle prior to the presenting injury. On physical examination, she was in mild distress with stable vital signs. Examination of the left ankle revealed intact skin and moderate soft tissue swelling. There was tenderness to palpation over the distal aspect of the left lateral malleolus and the calcaneofibular and anterior talofibular ligaments. There was mild tenderness over the medial malleolus, and no tenderness to the base of the fifth metatarsal. Passive range of motion was limited due to pain, and the patient could not bear weight on the left foot. Sensation was intact over the foot and ankle, and the pedal pulses were intact. The remainder of the physical exam was unremarkable. POCUS was performed in the ED. Longitudinal views of the left distal fibula demonstrated an obvious cortical defect of the left lateral malleolus consistent with fracture (Image 1). Plain radiographs of the left ankle, foot and lower leg were then obtained, which failed to demonstrate evidence of left fibular fracture. Due to the US findings suggestive of fracture, stress view radiographs of the left foot, ankle and leg were obtained with orthopedic consultation. These views demonstrated a non-displaced Weber B fracture of the left fibula (Image 2). Given this diagnosis, the patient was

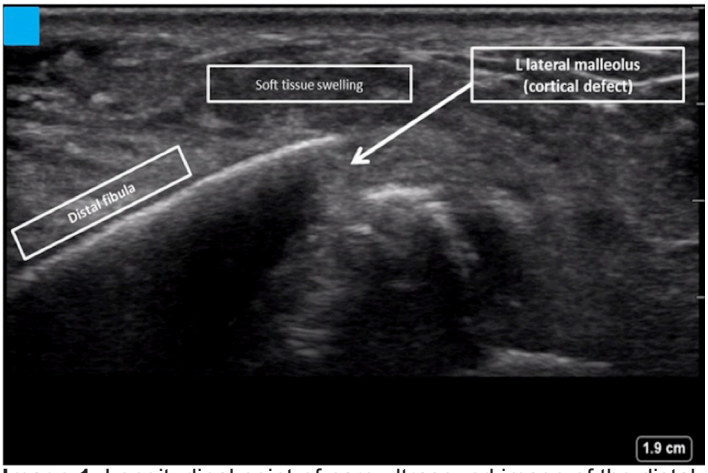

Image 1. Longitudinal point-of-care ultrasound image of the distal fibula demonstrating a cortical defect.

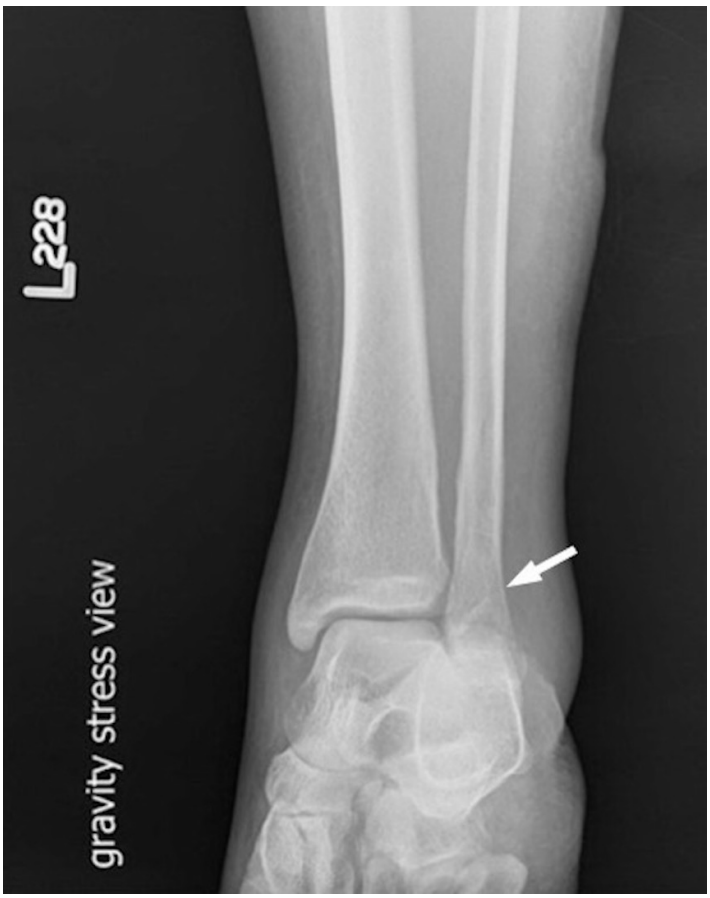

Image 2. Stress view radiography of the ankle demonstrating an irregularity of the lateral malleolus cortex consistent with a non-displaced Weber B fracture.

managed with a medical walking boot, acetaminophen/codeine for pain management and outpatient orthopedic follow up in 2-3 days. At follow up, orthopedics recommended that she continue use of the walking boot for 4-6 weeks.

DISCUSSION

While ankle injuries are a common presenting complaint in the ED, only 15% of undifferentiated patients presenting with ankle injury can be expected to have evidence of ankle fracture on plain radiographs.[1] Therefore, efforts have been made in recent years to rule-out ankle fracture by means other than radiography, most notably through the use of the OARs. These clinical decision rules have a high sensitivity, but a very low specificity, resulting in a large number of normal radiographs despite positive OAR findings.[1] However, the OARs have been independently validated as aiding emergency physicians in a faster diagnosis with fewer radiographs and decreased time in the ED for patients.[2]

When suggested by history and physical exam, the diagnosis of ankle fracture is most commonly made by plain radiography. However, plain radiography has an estimated sensitivity of 85.2% for lateral complex ankle fractures, suggesting a significant percentage of ankle fractures may go undiagnosed.[3] Additionally, 2014 meta-analysis suggested that after normal plain radiography the occult fracture rate may be as high as 24%.[4] Therefore, more advanced imaging, such as stress view radiography, computed tomography (CT) or magnetic resonance imaging (MRI), are warranted when the patient's history and physical exam are strongly suggestive of ankle fracture, even if initial plain radiography is normal.

As demonstrated in this case, POCUS may be used as an extension of the physical exam and provide critical information to ensure a diagnosis of fracture is not missed, especially when plain radiography is normal. Previous studies have analyzed cases in which ankle fracture was falsely ruled out by clinical exam or plain radiography but detected by POCUS, with detected occult fracture rates ranging from 8.96-14.1%.[3,5] Studies have estimated US to have a sensitivity of 87.3-100% and specificity of 93.1-99.1% for the evaluation of ankle fracture.[3,6,7] By one study, sensitivity is estimated to be 96.4% when radiography and US are used in combination to evaluate for ankle fracture, demonstrating the utility of US as a supplemental imaging modality.[3] US has additional utility in the pediatric population, where radiation exposure is especially concerning. The positive and negative likelihood ratios of US to diagnose ankle fracture in the pediatric population has been estimated to be between 9-20 and 0.04-0.08, respectively.[3] However, US is notoriously user-dependent, and generalizations about its diagnostic accuracy are difficult to make. Nevertheless, in the hands of a skilled user, US can provide important information in the evaluation of ankle fracture.

The Danis-Weber system is commonly used to characterize ankle fractures. This patient's fracture was consistent with a stable Type B fracture, which is defined as a spiral fracture of the lateral malleolus of the distal fibula,

beginning at the level of the joint and including a partial syndesmotic injury.[8] Type B fractures may be associated with a stable ankle joint. However, in the setting of a ruptured deltoid ligament, a Type B fracture is unstable.[9] Ankle stability is commonly determined using stress view radiography, where distal joint space clearing of 2mm or greater is indicative of an unstable joint.[8]

As in this case, ED management for a patient with a stable Type B fracture should include immobilization with a medical walking boot, weight bearing as tolerated, adequate pain control and referral to an orthopedic surgeon for surgical evaluation and follow up. Stress view radiography should be obtained to determine ankle stability, as unstable Type B fractures frequently require surgical fixation.

A missed or mischaracterized ankle fracture may have consequences on the future health of the patient. Inadequate immobilization may result in further damage, putting the patient at greater risk for the development of post-traumatic osteoarthritis, which accounts for 70% of all cases of ankle osteoarthritis.[10] POCUS can play a critical role in identifying and correctly characterizing ankle fractures.

POCUS is an important imaging modality in fracture assessment due to its availability at the patient's bedside, ease of use, and multiplanar diagnostic capabilities. Its usefulness includes injury assessment for fracture when radiographs are not immediately available, and detecting occult fractures not revealed on radiographs. Sonographic evaluation of bone, however, has limitations and should always be coupled with radiographs and possibly advanced imaging modalities such as CT and MRI when clinically indicated.

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The Neurocardiogenic Spectrum in Subarachnoid Hemorrhage: A Case Report and Review of the Literature

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A 36-year-old man was brought to our emergency department after successful resuscitation of out-of-hospital cardiac arrest with the whole spectrum of neurocardiogenic effects in subarachnoid hemorrhage: electrocardiographic changes, regional wall motion abnormalities, and elevations of cardiac enzymes. Coronary angiography revealed normal coronary arteries but showed the midventricular type of Takotsubo cardiomyopathy in the left ventriculography. Subsequently, cerebral computed tomography revealed diffuse subarachnoid hemorrhage and generalized cerebral edema with brain herniation. Brain death was diagnosed. This case highlights the possibility of an acute cerebral illness (especially subarachnoid hemorrhage) as an underlying cause of cardiac abnormalities mimicking myocardial ischemia. [Clin Pract Cases Emerg Med. 2017;1(1):16–21.]

INTRODUCTION

Subarachnoid hemorrhage accounts for 10% of hemorrhagic strokes, most of which are caused by ruptured saccular aneurysms with a mortality rate of up to 50%.[1] Most deaths occur within the first two days of onset, with the majority related to the initial hemorrhage.[2] Complications resulting from subarachnoid hemorrhage are rebleeding,[3] vasospasm and delayed cerebral ischemia,[4] hydrocephalus,[5] increased intracranial pressure,[6] seizures[7] and hyponatremia.[8] Cardiac abnormalities such as electrocardiographic changes, left ventricular dysfunction, and troponin elevations mimicking myocardial ischemia can also occur.

CASE REPORT

A 36-year-old man was brought to our emergency department (ED) by emergency medical services after witnessed loss of consciousness in a public toilet. No medical history regarding symptoms before collapse, significant health issues, or illicit or recreational drug use was known at ED arrival. When the paramedics arrived at the scene, the patient was in asystole. After 10 minutes of cardiopulmonary resuscitation the patient showed return of spontaneous circulation (ROSC) and was transported to our ED after intubation on scene.

In the ED, blood pressure was 85/40 mmHg, heart rate 120 bpm and oxygen saturation was 98% on mechanical ventilation (FiO2 100%). Heart sounds were regular with no murmurs and the lungs were clear to auscultation bilaterally. Glasgow Coma Scale was 3, pupils were dilated with minimal pupillary response and Babinski's reflex was negative bilaterally.

The electrocardiogram showed a narrow complex tachycardia with 150 bpm, ST-segment elevation in leads aVR, V1, V2 and ST-segment depression in leads II, III, aVF and V4 to V6 (Image 1).

Laboratory exams showed increased levels of high sensitive cardiac troponin T, creatine kinase and brain type natriuretic peptide. Suspecting fatal arrhythmia due to myocardial infarction as underlying cause, the patient was treated with aspirin and low molecular weight heparin. Acute coronary angiography was performed, which revealed normal coronary arteries, but demonstrated the midventricular type of Takotsubo cardiomyopathy in the left ventriculography. The left ventricular function was impaired with an ejection fraction of 47%. For further diagnostic workup, cerebral computed tomography with angiography was performed showing diffuse subarachnoid hemorrhage extending into the ventricular

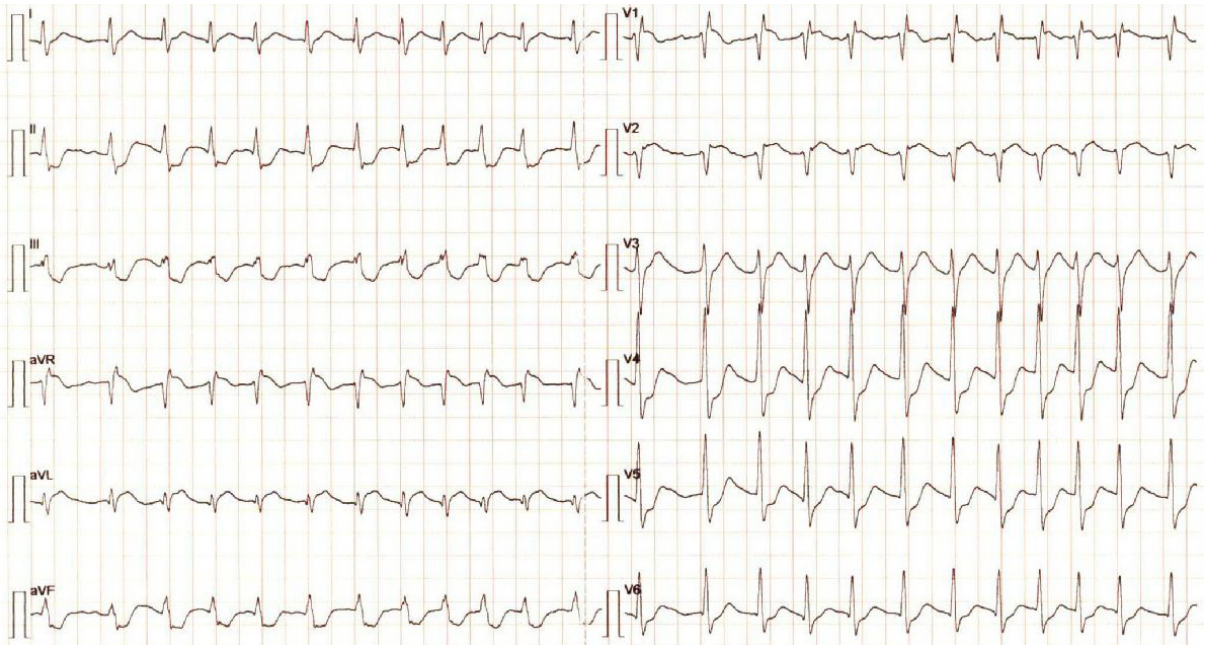

Image 1. Electrocardiogram showing a narrow complex tachycardia with 150 bpm, ST-segment elevation in leads aVR, V1, V2 and ST-segment depression in leads II, III, aVF and V4 to V6.
BPM, beats per minute

system due to a ruptured basilar artery aneurysm, and generalized cerebral edema with brain herniation and absent peripheral perfusion (Image 2).

According to his clinical presentation with persistent Glasgow Coma Scale of 3 without analgosedation and decerebrate posturing (adducted and extended arms with pronated wrists and flexed fingers, as well as extended legs with plantar flexion of the feet), the Hunt and Hess score was 5 (Table 1).

Because of persistent shock the patient was treated in the ED with epinephrine infusion with a maximum dose up to 0.3 mcg/kg/minute. Due to severe subarachnoid hemorrhage and loss of brainstem reflexes no additional intervention was recommended by neurosurgery. For further care the patient was transferred to our intensive care unit. Brain death was diagnosed by a neurologist and an intensive care physician who performed an apnea test. The family denied consent to organ donation.

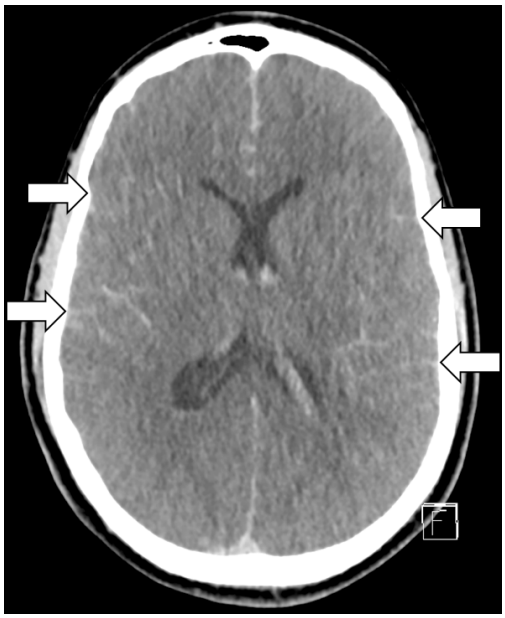

Image 2. Cerebral computed tomography, showing diffuse subarachnoid hemorrhage with generalized cerebral edema.

DISCUSSION

It is speculated that cardiac dysfunction after subarachnoid hemorrhage is most likely caused by centrally mediated release of catecholamines within the myocardium due to hypoperfusion of the posterior hypothalamus.[10-12] Histological analysis of myocardial tissue in patients after subarachnoid hemorrhage typically demonstrated subendocardial contraction band necrosis, also known as myocytolysis, without coagulation necrosis, as found in myocardial infarction.[13-20] Clinically, the neurocardiogenic effects of subarachnoid hemorrhage may present with electrocardiographic changes, elevations of troponin and/or brain type natriuretic peptide, as well as regional wall motion abnormalities, including Takotsubo cardiomyopathy.

Depending on the study, electrocardiographic abnormalities occur in 27% up to 100% of patients with subarachnoid hemorrhage.[21-25] The most striking electrocardiographic abnormalities are found within the first 48 to 72 hours,[21-22] which are summarized in Table 2.

ST-segment elevation typically is found in the precordial leads[34] and seems to occur mainly in those with apical and

Table 1. Hunt and Hess classification for grading patients with subarachnoid hemorrhage.[9]

Grade	Neurologic status
1	Asymptomatic, or minimal headache and slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
3	Drowsiness, confusion, or mild focal neurologic deficit
4	Stupor, moderate or severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances
5	Deep coma, decerebrate rigidity, moribund appearance

Table 2. Electrocardiographic findings in subarachnoid hemorrhage, modified from references.[26-33]

Morphological changes	Rhythm disturbances
peaked P-wave, short PR-interval	sinus bradycardia, sinus tachycardia
high R-wave	wandering atrial pacemaker, atrial fibrillation, atrial flutter
ST-segment elevation, ST-segment depression	atrioventricular block
QT-interval prolongation	premature atrial, junctional, ventricular complexes
deep symmetric T-wave inversion	ventricular tachycardia (including Torsades de Pointes)
prominent U-wave	

midventricular regional wall motion abnormalities.[35-36] QT-interval prolongation is more common with subarachnoid hemorrhage than with other forms of acute cerebrovascular disease and is responsible for the greater relative risk of ventricular tachyarrhythmia.[37-39] Therefore, constant electrocardiographic monitoring in the acute phase of subarachnoid haemorrhage is recommended.

The electrocardiographic changes are predominantly reflective of ischemic changes in the subendocardium of the left ventricle due to the release of large amounts of catecholamines. The diagnosis of myocardial injury in subarachnoid hemorrhage can be established by elevation of serum troponin, which can be observed in approximately 20% to 40% of patients, depending on the used troponin assay.[11,18,40-42] The elevation of troponin is usually mild to moderate and less pronounced than in myocardial infarction.[42] Elevated peak troponin levels are associated with an increased mortality and worse functional outcome.[43]

Elevated brain type natriuretic peptide levels can be detected after subarachnoid haemorrhage as well,[44-46] probably due to hypoxia of the hypothalamus, endothelin-1 release, and excess catecholamine secretion, which increases the afterload on the cardiac ventricles.[47,48] Elevated brain type natriuretic peptide levels have been associated with impaired left ventricular function,[49] cerebral vasospasm and delayed ischemic neurological deficits,[50] as well as increased mortality.[51]

Acute left ventricular systolic dysfunction is a well-recognized complication after subarachnoid haemorrhage,[20,52-56] occurring in up to 30% of patients. This has been referred to as neurogenic stress cardiomyopathy or neurogenic stunned myocardium.[20] Left ventricular systolic dysfunction usually develops within the first two days after subarachnoid hemorrhage. The timing of recovery of left ventricular systolic dysfunction ranges from a few days to weeks.[53,56-61] Independent predictors of neurogenic stunned myocardium after subarachnoid hemorrhage include severity of neurological injury, troponin, creatine kinase-MB and brain type natriuretic peptide elevation as well as female gender.[11,20,52] Patients with neurogenic stunned myocardium are at particularly high risk for potentially fatal complications such as ventricular arrhythmias.[33] Furthermore, neurogenic stunned myocardium is associated with an increased risk of subarachnoid hemorrhage-associated cerebral vasospasm. It is likely that the alteration in cerebral perfusion associated with subarachnoid hemorrhage, combined with impaired left ventricular systolic dysfunction, contributes to vasospasm severity.[62]

The most common wall motion abnormalities in neurogenic stunned myocardium due to subarachnoid hemorrhage appear to be either global hypokinesis or the basal and midventricular type of Takotsubo cardiomyopathy. The classical apical type Takotsubo cardiomyopathy is less common.[35,36,53,54,56,63] The exact reason for the different regional wall motion abnormalities in neurogenic stunned myocardium is not known. It might be explained by the unequal distribution of adrenergic receptors in myocardial cells, adrenergic receptor polymorphisms, and variations in individual susceptibilities to the circulating catecholamines in certain regions of the heart. Areas with higher density of adrenergic receptors may determine the area of hypokinesis.[64-66]

In our patient, neurogenic stress cardiomyopathy was difficult to distinguish from acute myocardial infarction given the associated electrocardiogram changes. Potential indicators that favor a diagnosis of neurogenic stunned myocardium include wall motion abnormalities disconcordant with a single

epicardial coronary distribution and a relatively minor cardiac troponin release relative to the magnitude of left ventricular dysfunction.[41] Despite these features, coronary angiography may be required in the acute setting to distinguish between neurogenic stunned myocardium and acute myocardial infarction in some patients, especially those after cardiac arrest as our patient.

Clinical management of patients with neurogenic stunned myocardium is generally supportive because left ventricular function usually normalizes spontaneously within a few days to weeks.[53,56-61] It is important to exclude dynamic left ventricular outflow tract obstruction with echocardiography in patients with severe heart failure or significant hypotension.[67,68] Whenever possible, the precipitating cause of neurogenic stunned myocardium should be addressed. In the absence of contraindications, β-blockers should be considered early to temper the catecholamine surge, and angiotensin converting enzyme inhibitors should be given until left ventricular function completely recovers. Diuretics are effective in most cases of congestive heart failure. Patients with cardiogenic shock should be treated with standard therapies including inotropes (in the absence of dynamic left ventricular outflow tract obstruction) and mechanical ventilation as needed.[69]

CONCLUSION

This case highlights the potential severity of the cardiac manifestations of subarachnoid hemorrhage as well as the need to consider a cerebral illness (especially subarachnoid hemorrhage) as a cause of electrocardiographic changes suggestive of myocardial infarction, troponin elevations and regional wall motion abnormalities, in order to avoid possible inappropriate or delayed therapy. In cases with absent clinical context of acute coronary syndrome, or a history of thunderclap headache, potentially harmful antiplatelet and anticoagulation therapies should be delayed until coronary artery disease involvement has been proven.

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Pasteurella multocida Epiglottitis

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Epiglottitis is an uncommon but life-threatening disease. While the most common infectious causes are the typical respiratory pathogens, *Pasteurella multocida* is a rare causative organism. We present a case of *P. multocida* epiglottitis diagnosed by blood culture. The patient required intubation but was successfully treated medically. *P. multocida* is a rare cause of epiglottitis; this is the ninth reported case in the literature. Most diagnoses are made from blood culture and patients usually have an exposure to animals. [Clin Pract Cases Emerg Med. 2017;1(1):22–24.]

INTRODUCTION

Epiglottitis is characterized by inflammation of the epiglottis and surrounding structures and classically presents with severe sore throat, dysphagia, and leukocytosis.[1] Although uncommon, it is a life-threatening disease that can cause acute airway obstruction. The most common etiology of epiglottitis is an infection by *Streptococcus pneumonia*, *Staphylococcus aureus,* or *Haemophilus influenzae* (type B, A, F, and nontypable), while other bacteria, viruses, and fungi constitute more rare forms.[2,3,17-19]

Pasteurella multocida is a Gram-negative coccobacilus that colonizes the respiratory system of dogs and cats, as well as commercial livestock and wild animals.[4] In cows *P. multocida* is a leading cause of bacterial pneumonia and morbidity known as bovine respiratory disease.[10] In humans, there are only eight reported cases of *P. multocida* epiglottitis in the literature since 1977.[1,2, 4-9] All eight reports involve middle-aged adults, seven of whom were men. Three cases involved immunosuppressed patients.[1,4,5] Seven cases involved contact with animals, generally domesticated cats.[1,2,3-7,9] Pasteurella was diagnosed as the causative agent by blood culture in seven cases, with the remaining case by post-mortem tissue culture.[1,2,4-9] We present a case of *P. multocida* epiglottitis involving a healthy middle-aged woman who had two pet dogs but could not recall any bites, scratches, or inoculations.

CASE REPORT

A 49-year-old female with a past medical history of hypothyroidism, prurigo nodularis, and depression presented to our emergency department (ED) with one day of gradual onset of sore throat, dysphagia, odynophagia, and chills. She was very concerned because she had the sensation that she could not swallow her own saliva. On exam she was non-toxic appearing, afebrile, and had normal vital signs. Her voice and oropharyngeal exam were normal. Her external neck was notable for mild left submandibular lymphadenopathy. The rest of her exam was normal. She did not reliably take her levothyroxine at home for her hypothyroidism, she had no known allergies, no known sick contacts, no recent travel, nor did she use illicit drugs; her immunizations were up to date.

She was given a dose of ketorolac and observed. On re-evaluation she was unable to tolerate any liquids by mouth. Her oropharynx and larynx were then topicalized with lidocaine and a flexible fiberoptic endoscope was inserted orally to evaluate the larynx. The patient had a beefy, red, and edematous epiglottis with edema and erythema extending to the aryepiglottic folds and arytenoids, and she had mild pooling of secretions. The true vocal folds were symmetric and mobile without erythema or edema. An image from the initial laryngoscopy can be seen in Image 1. The patient was administered dexamethasone, ampicillin-sulbactam, diphenhydramine, and racemic epinephrine nebulizers. Her complete blood count was notable for a white blood cell count of 22.6 cells per microliter with a 91% neutrophil predominance, and two sets of blood cultures were taken. The otorhinolaryngologist we consulted performed a nasopharyngolaryngoscopy and recommended medical

management in the intensive care unit (ICU).

Several hours after admission to the medical ICU the patient's respiratory efforts increased, and she was taken to the operating room where the anesthesiologist performed an awake nasotracheal fiberoptic intubation with the acute care surgery team at the bedside for a potential emergent surgical airway. The patient was successfully intubated nasotracheally and returned to the ICU. The blood cultures drawn in the ED returned positive for pleomorphic Gram-negative rods on the first hospital day, which were later speciated for pan-sensitive *P. multocida*. A viral respiratory panel from the nasopharynx returned negative for adenovirus, respiratory syncytial virus, parainfluenza virus types 1, 2, and 3, as well as influenza viruses A and B.

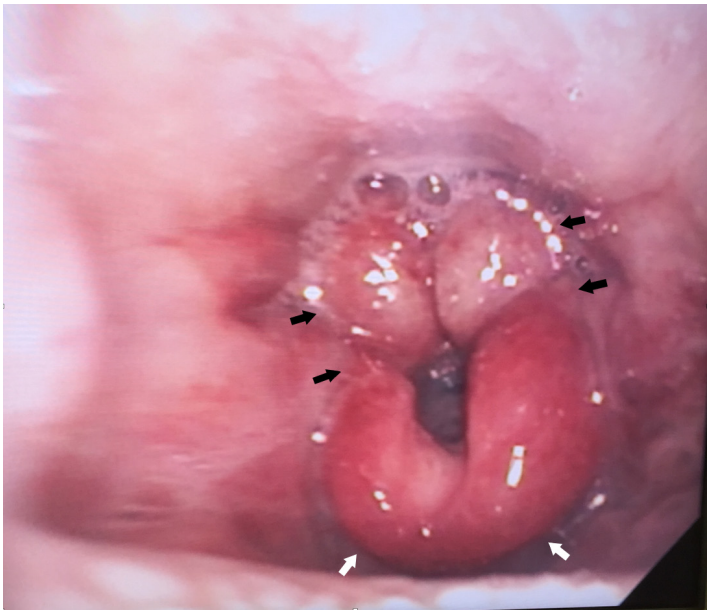

Image. Emergency department endoscopy demonstrating epiglottitis (white arrows), swelling of the aryepiglottic structures (black arrows), and pooling of secretions.

The patient had a midline catheter placed for home antibiotic infusions, and her antibiotics were tailored to ceftriaxone monotherapy. On hospital day three, laryngoscopy showed much improvement of her epiglottitis and the patient was successfully extubated. She was transferred to the medical-surgical floor on hospital day four, and then discharged home on hospital day eight with a plan for continued antibiotic infusion. During her hospitalization the infectious disease (ID) service was consulted for treatment recommendations. The ID team recorded a history of the patient owning a Chihuahua and a terrier at her home, though she could not recall any recent scratches, bites, or contact with animal saliva or mucous membranes. The patient followed up in otorhinolaryngology clinic one month later and had a repeat laryngoscopy, which did not show any evidence of epiglottitis.

DISCUSSION

Epiglottitis caused by *P. multocida* is rare; only eight other cases have been reported in the literature since 1977.[1, 2, 4-9] *P. multocida* is a Gram-negative coccobacillus commonly found in the upper respiratory tracts of animals, including domestic dogs and cats.[4] Exposure to animals was a common theme in previously reported cases of epiglottitis secondary to *P. multocida*. Patients with immunosuppression may have a greater susceptibility to infection.[1,11] Other risk factors for epiglottitis include lack of immunization and smoking.[11] *Haemophilus influenza* type B (*HiB*) used to be the most prevalent organism, but its incidence declined dramatically after the introduction of the pediatric vaccination series. Currently group A beta-hemolytic streptococci is the most common causative bacterial organism, and pediatric epiglottitis is less common than adult-onset epiglottitis.[12]

Our patient was an otherwise healthy 49-year-old woman without any significant co-morbidities that would suggest immunosuppression. She did have regular exposure to her two dogs, and it is possible that oral secretions from her dogs were the source of the infection; however, the exact mechanism of exposure was not confirmed.

The clinical presentation of adult patients with epiglottitis most commonly involves the rapid onset of sore throat with dysphagia, odynophagia and the inability to swallow secretions. [1,13] A normal oropharyngeal exam does not exclude epiglottitis. Additional signs may include a tripod patient positioning, cervical lymphadenopathy, and fever.[13, 14] Laboratory results often show a leukocytosis.[1] The causative organism can be confirmed with epiglottic tissue and blood cultures.[1] Treatment with penicillins, cephalosphorins, or fluoroquinolones is recommended for 1-6 weeks, depending on the severity of infection.[1,5] Steroids, racemic epinephrine and beta-agonists may also be considered, although they have not been proven to improve outcomes.[13]

Epiglottitis is suggested on lateral neck radiographs that demonstrate a "thumb sign," which indicates increased epiglottic and aryepiglotticwidth.[13,15] The epiglottis is typically 3-5 mm thick on a lateral neck radiograph.[13] The "vallecula sign" may also be seen on the lateral radiograph, in which a V-shaped space is observed extending from the base of the tongue to the epiglottis instead of a normal linear space.[13] However, one study showed that lateral radiography only had a sensitivity of 38% and specificity of 76% for diagnosing epiglottitis.[16] An ultrasound of the neck can also demonstrate an increased anteroposterior diameter of the epiglottis in epiglottitis and may aid in diagnosis.[17] Computed tomography is very accurate at diagnosing epiglottitis.[21] Suspected epiglottitis is probably best evaluated by direct visualization of the epiglottis.

Airway management is the cornerstone of therapy for severe cases and milder cases with poor trajectory. Intubating

a patient with epiglottitis (or suspected epiglottitis) should be presumed to be very challenging, and an awake intubation with simultaneous preparation for a possible surgical airway is likely the most prudent option; a surgical airway may be inevitable. The airway may need to be managed in the ED, but transferring the patient to the operating room with anesthesia and surgical backup may be more prudent.

Emergency physicians should be aware of this diagnosis because epiglottitis is a life-threatening disease. *P. multocida* epiglottitis is rarely the pathogen responsible, though diagnosis can be made by blood culture. Epiglottitis is rare in adulthood, but sore throat, dysphagia and odynophagia are common chief complaints. If a patient reports that they cannot tolerate their own secretions, further investigation is warranted.

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Spontaneous Bilateral Internal Carotid Artery Dissections in a Young Female with Headache

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Spontaneous cervical artery dissection (sCAD) occurs when the intimal lining separates from the outer wall of the artery. Although rare, it is a common cause of stroke in young people. Presentations range from isolated headache to severe stroke symptoms. A 41-year-old woman with minimal past medical history presented with left-sided headache and transient right leg weakness and numbness. The patient underwent computed tomography (CT) angiography of the neck that showed bilateral internal carotid artery dissections with a relative stenosis from pseudoaneurysm formation on the left. She was placed on a heparin drip and transitioned to warfarin but subsequently required stent placement 10 days later. If this patient had not undergone CT angiography at the time of presentation, she might have suffered significant morbidity and possible mortality. [Clin Pract Cases Emerg Med. 2017;1(1):25–27.]

INTRODUCTION

Spontaneous cervical arterial dissection (sCAD) is a rare but clinically important entity as it causes a significant proportion of strokes in young people. Dissection is found to be the underlying etiology of stroke in 0.4 to 4% of patients but as high as 20% in stroke patients younger than 30 years of age.[1] The pathophysiology of sCAD involves a tear in the intimal lining of the artery that leads to luminal stenosis or occlusion, intra-arterial thrombus formation, and ultimately stroke via either hypoperfusion or thromboembolism. The carotid or vertebral arteries can be affected, with the former being twice as common as the latter.[2] The total incidence of spontaneous internal carotid artery dissection (sICAD) is estimated at around three per 100,000,[3] but the exact incidence is unknown as presentations can range from asymptomatic to isolated headache or neck pain to transient ischemic attack to stroke. The incidence of bilateral carotid dissections is considered rare and is not fully known, but may be as high as 22% of sICADs.[4,5] We present a case of bilateral sICAD presenting with unilateral headache and blurry vision.

CASE REPORT

A 41-year-old female presented to the emergency department (ED) with four hours of severe sudden-onset left-sided retro-orbital headache. It was associated with blurry vision in her left eye and did not change in intensity. She initially delayed seeking medical evaluation but came to the ED when her symptoms persisted. After the headache had been present for two hours, she noted some altered sensation throughout her right lower extremity but had no complaints of focal weakness. Her medical history was only significant for depression and daily tobacco use. She did not have a history of headaches and there was no trauma.

The patient was evaluated by a physician at triage upon initial arrival in the department and was not noted to have any focal deficits. Her initial vital signs were only notable for a blood pressure of 125/95mmHg. She was evaluated by the treating physicians approximately 20 minutes after arrival and noted to have an NIH stroke scale score of three for slight flattening of the left nasolabial fold, drift in the right leg, and altered sensation in the right leg. Sensation was tested by comparing the perception of sharp stimuli between each lower extremity, which was reported to be more dull throughout the right lower extremity compared to the left in a non-dermatomal pattern. The patient underwent an emergent non-contrast computed tomography (CT) of the head followed by CT angiograms of the neck and brain. The on-call neurologist was consulted and noted no focal deficits on exam

with an NIH stroke scale of zero approximately 40 minutes after the exam by the emergency physician. Her imaging studies were reviewed and she was found to have bilateral internal carotid artery dissections with pseudoaneurysm formation on the left creating a relative stenosis (Image).

After discussion with the neurosurgical service, she was started on a heparin drip and admitted to the ICU for monitoring. She underwent cerebral angiography but did not require any intervention at the time, as good flow was seen distal to the dissections and pseudoaneurysm. She was discharged home three days later on an enoxaparin bridge to warfarin.

She re-presented to the ED 10 days later with right-sided numbness that developed into aphasia and right-sided weakness. Her initial complaint was tingling on the right side of her face and along the right arm but this progressed into severe aphasia, dysarthria, diminished strength throughout the right upper and lower extremities, and decreased response to painful stimuli on the right side while she was in the ED. She was taken back to the angiography suite and a stent was placed in the left internal carotid artery. She was discharged three days later on aspirin and clopidogrel. A follow-up CT angiogram of the neck performed four weeks later showed a stable right internal carotid artery dissection and patent left-sided carotid stent.

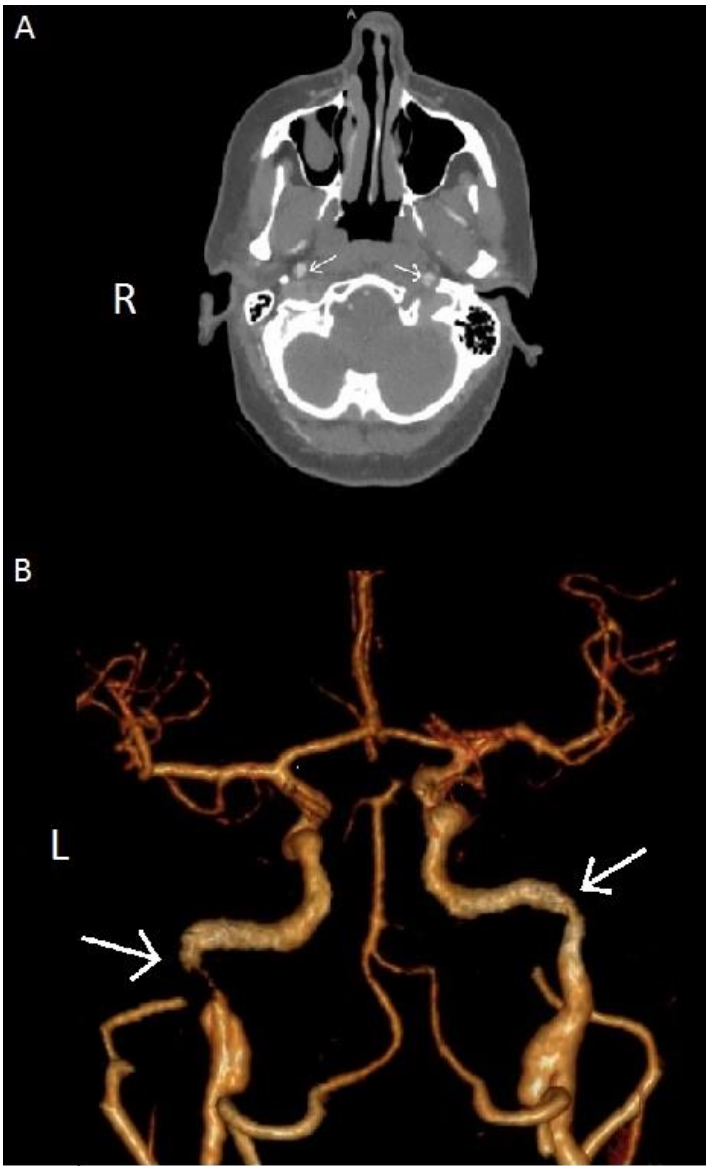

Image. A. Axial CT angiography. Arrows mark intimal flaps in right and left internal carotid arteries. B. 3D reconstruction of CT angiography of the brain with luminal narrowing of bilateral internal carotid arteries marked by arrows. *CT,* computed tomography

DISCUSSION

sCADs cause a large proportion of ischemic strokes in young people, especially in those without known cardiovascular risk factors, with an average age of 45.[6] Reported incidence of sCAD is likely underestimated as some patients can present with minimal or even no clinical signs.[5–8] In fact, the most common presenting symptom is unilateral frontal headache.[9] As such, many patients with headache may not undergo CT angiography to evaluate for dissections. Although the diagnosis is difficult because of the variation in presentation, it is a crucial diagnosis to make in the ED with estimates of long-term neurologic sequelae of 30% and mortality of up to 2%.[2]

In the case presented above, the patient's focal neurologic signs were transient and were not identified on two of her three examinations, including by the attending neurologist, which highlights how easily this diagnosis can be missed. A study by Lee et. al. found transient ischemic attack (TIA) in 23% of sCADs and stroke in 56%.[6] Associated risk factors for sCAD include smoking, migraines, and fibromuscular dysplasia (FMD).[6] Horner syndrome is found in 25% of patients with sCAD[6] and is slightly more common when pseudoaneurysm is present. Hassan et. al. found pseudoaneurysms in 90% of multiple dissections.[5]

There is little in the medical literature about multiple cervical artery dissections. The incidence ranges from 13-22% of all sCADs, but these studies are limited to small sample sizes.[5,6] Signs and symptoms of bilateral sCADs can range from stroke to TIA to only headache, making diagnosis difficult.[8,10] Either or both sides may be symptomatic. Our patient was only symptomatic of her left-sided dissection at the time of presentation. Diagnosis is confirmed with imaging, which can include ultrasound, magnetic resonance imaging or magnetic resonance angiography, or CT angiography.[11]

Management typically includes anticoagulation or antiplatelet therapy, with endovascular intervention having emerged as an additional or alternative therapeutic option.[8,10,12–14] The Cervical Artery Dissection in Stroke Study (CADISS), published in 2015, compared the use of antiplatelet therapy and anticoagulation in the long-term treatment of stroke secondary to CAD and found no significant difference between the two. Antiplatelet regimens were not uniform

and consisted of aspirin, dipyridamole, or clopidogrel, alone or in combination.[13] No definitive guidelines exist regarding the role of endovascular therapy, but a systematic review out of China suggested stent therapy for patients with recurrent symptoms despite adequate medical therapy, significant cerebral hypoperfusion, a symptomatic or expanding pseudoaneurysm, or a contraindication to anticoagulation.[14] Since there is no consensus on medical versus endovascular treatments or even a specific ideal medical regimen, current practices will likely vary and rely on discussions between emergency physicians, specialists, and patients. Our patient was managed with stenting on her return visit after previously being treated with anticoagulation, consistent with the current literature.

CONCLUSION

In conclusion, sCAD is a rare but potentially deadly phenomenon that needs to be considered in patients with headache and neck pain, particularly in the presence of neurologic symptoms. Bilateral dissections, in particular, carry increased risk by making larger regions of the brain susceptible to ischemia via occlusion and thromboemboli. CT angiography does not need to be performed on all patients with head or neck pain, but should be performed when focal neurological complaints or findings are noted and strongly considered when patients report neurologic symptoms prior to arrival.

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Salmonella Urinary Tract Infection Heralding Thoracic Mycotic Aneurysm: Case Report as Medical Apology

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We report a case as a patient apology as a means of teaching other physicians about a unique presentation of a rare disease. *Salmonella* species are unusually isolated organisms in urine. In the case described, appreciation for the rarity of *Salmonella* species in the urine facilitated recognition of a serious disseminated *Salmonella* infection. Physicians should consider disseminated *Salmonella* infection, as was found in a patient with an aortic mycotic aneurysm, after isolation of *Salmonella* in urine despite an initially benign clinical presentation. [Clin Pract Cases Emerg Med. 2017;1(1):28–30.]

CASE REPORT

A 71-year-old man presented to an outside emergency department (ED) with chief concerns of abdominal pain and anorexia that had been present for four weeks. Findings of an extensive work-up were subsequently unrevealing, including normal results of colonoscopy, esophagogastroduodenoscopy, colon biopsy, mesenteric Doppler studies, and computed tomography (CT) of the abdomen and pelvis. Video capsule endoscopy demonstrated a questionable adynamic ileus. Treatment with dicyclomine hydrochloride, polyethylene glycol 3350, tramadol, and simethicone had failed to relieve symptoms. The patient's family decided to sign him out of the outside hospital from the inpatient service and drive him directly to our ED for evaluation in the middle of the night.

On initial presentation to our ED, the patient complained of left-sided abdominal pain described as aching and one episode of hematuria. Past medical history included osteoarthritis, hypertension, chronic obstructive pulmonary disease, coronary artery disease, nonischemic cardiomyopathy, chronic atrial fibrillation, embolic stroke, and mediastinal thymoma with sternotomy and subsequent pleural effusion and empyema. Initial findings were as follows: temperature 36.6°C oral, blood pressure 142/95 mm Hg, pulse 106 beats per minute, oxygen saturation 98% while breathing room air, and respiratory rate of 16 breaths per minute. Physical examination findings and results of laboratory analysis were reassuring except for an international normalized ratio of 5.5 and evidence of hemoconcentration. The patient was given intravenous (IV) fluids and 1,000 mg of acetaminophen and was then dismissed. Results of urine culture were positive for growth of more than 100,000 colony-forming units of *Salmonella* species 72 hours later.

Per protocol, the positive urine culture results were flagged and presented to the physician working clinically. On the basis of the patient's prior presentation and the rarity of *Salmonella* in the urine, the patient was asked to return immediately for reevaluation. On return to the ED, the patient's wife expressed sincere confusion and disappointment as she tried to explain how her husband, a hardworking man, was told to "walk it off" and that his irritable bowel syndrome was "just something he had to learn to live with." The patient appeared pale and acutely ill with closed eyes and persistent moaning. Findings at presentation were temperature 37.2°C oral, blood pressure 102/55 mm Hg, pulse 93 beats per minute, oxygen saturation 95% while breathing room air, and respiratory rate of 22 breaths per minute. Pain was rated as 10 on a scale of 0 to 10. Positive physical examination findings included flat neck veins, dry mucous membranes, healed scars on the chest, and left lower quadrant abdominal pain without tenderness or peritoneal signs, inguinal tenderness, or

adenopathy. Sepsis work-up and blood cultures were ordered, as was CT because of concern for possible disseminated infection. The patient was given 1 gram of ceftriaxone, IV fluids, and hydromorphone.

CT showed a contained pseudoaneurysm in the descending thoracic aorta (4.7×4.0×5.7 cm) about 2 cm in diameter from the native aortic wall to the outer edge of the outpouching; it had a bilobed appearance and a total craniocaudal measurement of 2.6 cm for both outpouchings (Images 1 and 2). The inferior-most origin of the pseudoaneurysm was noted about 3 cm above the origin of the celiac artery. Surrounding hematoma was seen on the descending thoracic aorta with inflammation and "old" hematoma settling down into the diaphragm.

The patient was admitted to a vascular surgery service, and IV ceftriaxone therapy was continued before surgical resection and replacement of a segment of the descending thoracic aorta with a cryopreserved aortic interposition graft. After a two-week hospital stay, the patient was dismissed receiving outpatient IV antibiotic infusion therapy.

DISCUSSION

Salmonella is an unusual organism in the urine; in fact, it accounts for less than 0.07% of all isolates.[1] *Salmonella* organisms isolated from the urine can result from fecal contamination. A previous case series of urinary tract infections with *Salmonella* isolation suggested that urinary tract infections caused by *Salmonella* isolation in the urine did not substantially differ from typical urinary tract infections except that they could be more severe in patients who had received renal transplants.[2] However, additional case reports have primarily been related to the association between *Salmonella* bacteriuria and urinary tract anatomical malformations or have suggested the presence of other serious invasive illness.[3,4]

In the case described, the patient had salmonellosis with bacteremic seeding of the aorta (aortitis) and persistent bacteremia. The anorexia, left lower quadrant pain, and generalized lethargy that ensued over weeks resulted from persistent bacteremia and also explained his urinary tract infection. Mycotic aneurysm is a misnomer because it is often the result of bacteria. *Salmonella* infection is a common cause of a mycotic aneurysm; however, the thoracic aorta is an uncommon location for a pseudoaneurysm.[5] Other organisms classically associated with mycotic organisms include *Mycobacterium tuberculosis* and *Treponema pallidum;* however, with routine antibiotic use *Staphylococcus aureus*, *Streptococcus* species, and *Enterococcus* species have become more common.[6] Fever, anorexia, and chills are common presentations. Left lower quadrant pain is not as common, and a positive result on urine culture prompting a routine callback in a busy ED is most definitely an uncommon presentation for an uncommon disease.

The patient had a very thorough work-up preceding the final diagnosis, including a CT of his chest and the CT of his abdomen and pelvis. However, because the images did not include the area of pseudoaneurysm, his final diagnosis remained unclear until the urine culture provided additional justification to repeat imaging. The time to diagnosis is understandable as this was a rare presentation of a rare disease that was discovered only when *Salmonella* grew where it did not belong.

Difficulty in diagnosing mycotic aneurysms accounts for the high mortality and is due to the vagueness of the initial

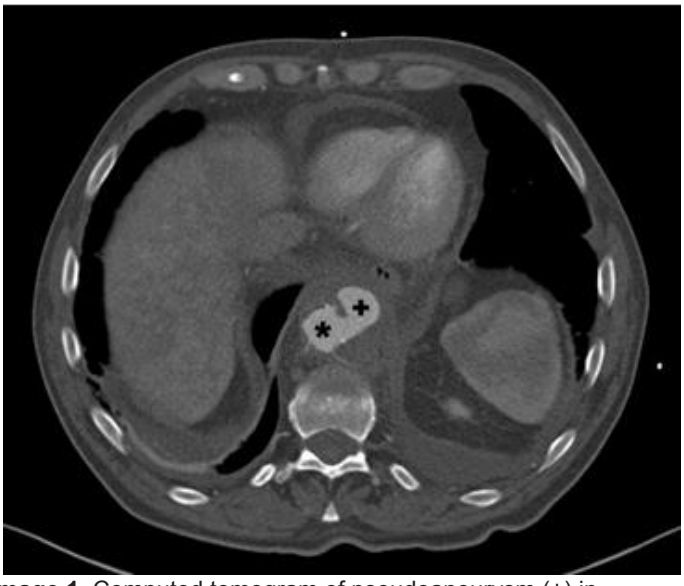

Image 1. Computed tomogram of pseudoaneurysm (+) in descending thoracic aorta (*).

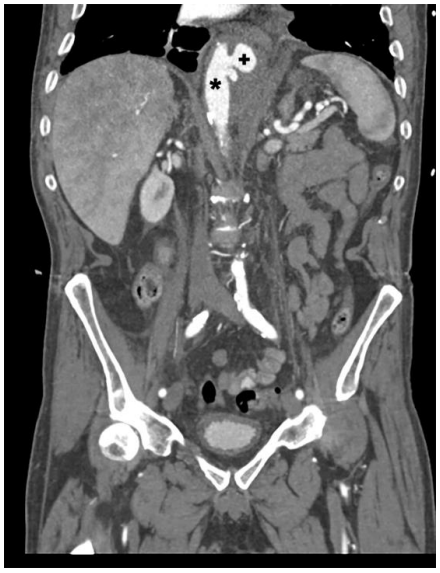

Image 2. Coronal computed tomogram of pseudoaneurysm (+) in descending thoracic aorta (*).

clinical presentation, as was the case with the patient described. Commonly, patients report days to months of intermittent fevers, fatigue, abdominal pain, nausea, anorexia, or back pain.[5] Given the rarity of infected aortic aneurysms, case report data describe only standard diagnostic techniques, including CT, echocardiography, and magnetic resonance imaging. Transesophageal echocardiography better delineates thickening of the aortic wall or sequela of dissection.[7] If targeted antibiotic therapy can occur before aneurysm formation, then mortality may be as low as 25%.[5] However, if no diagnosis is made until the time of aneurysmal rupture, mortality may exceed 65%.[6] Historically, mortality rates with medical treatment alone may reach 100%. Therefore, experts agree that treatment should involve surgical replacement of the infected aorta in conjunction with targeted antibiotic therapy.[8]

ADDENDUM

This case report is offered to the family as an apology for the patient's delayed diagnosis and a promise to share our knowledge with the medical community to prevent missing this rare diagnosis for future patients. The family was intensely gratified and comforted that our reporting the case might afford a quicker diagnosis for a patient who presents in the same manner.

An apology is a restoration of trust that offers a continued partnership in care. Apologies heal by satisfying one or more of the following values: restoration of self-respect, assurance of shared values, elimination of self-blame, preservation of safety in a relationship, continuing dialogue, and reparation of harm.[9] Our apology eliminated self-blame for the family, assured shared values and safety in the relationship, and made reparations through commitment between the patient and family and the healthcare providers to broaden dissemination of education with hopes of care for other patients.

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Traumatic Pneumothorax Following Acupuncture: A Case Series

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Acupuncture and dry needling are used by a range of health professionals to treat conditions such as musculoskeletal pain. Treatment occurs both in an outpatient setting and in emergency departments (ED). Acupuncture and dry needling are considered to be relatively safe techniques with a low risk of serious adverse events. We report three cases of traumatic pneumothorax following acupuncture/dry needling that presented to our ED between 2014 and 2016. [Clin Pract Cases Emerg Med. 2017;1(1):31–32.]

INTRODUCTION

Acupuncture is a popular alternative medicine technique that has been practiced in China for over 3,000 years. It involves the placement of solid needles of 10 – 100 mm length through the skin. Depth of insertion varies from a few mm to several cm, often into muscle tissue. For musculoskeletal (MSK) conditions, needles of 0.2 – 0.35 mm diameter are typically placed 10 – 65 mm deep into tissue and left *in situ* for 10 -30 min.[1] It has been estimated from post-mortem examinations that a puncture depth of 10 – 20 mm is sufficient to reach the lungs.[2] We report three cases of traumatic pneumothorax following acupuncture/dry needling that presented to our hospital. All cases involved young females of low body mass index (BMI) who underwent acupuncture/dry needling to the shoulder and upper back region of shoulder for MSK pain.

CASE REPORTS

Case 1

A 24-year-old female with a BMI of 22 had dry needling to her left posterior shoulder for MSK pain; 30 minutes later she developed severe pain around the site and shortness of breath. She presented to the ED five hours later with a respiration rate of 22 breaths per minute (BPM) and SpO_2 of 98% on room air. Her pain score was 7/10 on a numeric rating scale (NRS). Reduced air entry over the left anterior chest was noted by auscultation. A chest radiograph (CXR) revealed a 22% left apical pneumothorax (calculated by interpleural distance).[3] The patient was observed overnight and discharged after a repeat CXR the following morning showed no change of the pneumothorax size. A repeat CXR 10 days later showed full resolution.

Case 2

A 21-year-old female with a BMI of 18 developed severe, right-sided chest pain and shortness of breath shortly after receiving acupuncture for "knots" in her neck. She presented to the ED the following day with 7/10 NRS right-sided pleuritic chest pain. Her vital signs were a heart rate of 102, a respiration rate of 22 BPM and SpO_2 of 100% on room air. Decreased breath sounds were noted over the right upper zone. A CXR demonstrated a 26.3% right-sided pneumothorax.[3] The patient was discharged home and re-presented the following day for a repeat CXR, which demonstrated no change in pneumothorax size. On day four after her initial presentation, the patient re-presented with sudden onset of worsening shortness of breath and pain. A CXR was taken and showed that the pneumothorax was resolving. She was given stronger analgesia, which improved her symptoms. A further repeat CXR at nine days showed full resolution of the pneumothorax.

Case 3

A 21-year-old female with a BMI of 21 presented with

6/10 (NRS) sharp left-sided chest pain, dizziness, nausea and pre-syncope one-and-a-half hours after dry needling for left-sided MSK shoulder pain. Her vital signs were a heart rate of 66, a respiration rate of 24 BPM and SpO_2 of 100% on room air. Decreased breath sounds were noted over the left lung field. A CXR demonstrated a 16.2% left apical pneumothorax.[3] A repeat CXR four hours later showed a 20.6% pneumothorax.[3] Since the patient remained clinically stable, she was managed conservatively with a follow-up appointment three days later. Here, CXR showed that the pneumothorax was resolving. At a further appointment 21 days after the initial presentation, CXR demonstrated full resolution.

DISCUSSION

Health professionals including ED clinicians use acupuncture/dry needling.[4] It is generally considered to be low risk. Serious complications such as pneumothorax, subarachnoid hemorrhage, nerve lesions, infection and retained needles have been reported in the literature but are thought to be rare.[5,6] A large prospective observational study assessing the safety of acupuncture in Germany [7] and a review of safety data from the British National Health Service found an incidence of pneumothorax of less than one in a million per procedure.[8,9] Since these studies included all applications of acupuncture, for example including to the knee, the incidence of pneumothorax due to acupuncture to the chest wall is likely to be higher. Our report of three cases in as many years supports that notion. Since all cases occurred in young women of low BMI we further hypothesize that this patient population is at increased risk.

CONCLUSION

Traumatic pneumothorax is an important differential in the setting of dyspnea with recent acupuncture/dry needling. Patients with a low BMI may be at particular risk. Furthermore, patients should be informed of this uncommon but serious complication before undergoing acupuncture/dry needling.

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A Case of Severe Accidental Hypothermia Successfully Treated with Cardiopulmonary Bypass

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After missing for seven days, a 34-year-old female was found with a rectal temperature of 19.8oC. Instead of attempting aggressive rewarming in the emergency department she was directly transferred to the operating room for extracorporeal rewarming. She received cardiopulmonary bypass (CPB) for 66 minutes at an initial warming rate of 12oC/ hour and warmed to 36.2oC. Her postoperative course was complicated by sepsis, which eventually led to bilateral below-knee amputations after refusing antibiotics. She was discharged 22 days after admission, with full neurologic recovery. This remarkable case highlights the emerging role of CPB as the definitive therapy for severe accidental hypothermia. [Clin Pract Cases Emerg Med. 2017;1(1):33–36.]

INTRODUCTION

Hypothermia is defined as a decrease in core body temperature, broadly classified as mild (32-35 °C), moderate (28-32 °C), and severe (< 28 °C).[1] The spectrum of treatments is equally broad. At the mild end of the spectrum, peripheral rewarming strategies such as the application of warming packs to the groin and axillae are adequate. However, severe hypothermia requires invasive central rewarming interventions.[2] This case report features a patient who presented with severe accidental hypothermia and her ensuing management using cardiopulmonary bypass.

CASE PRESENTATION

A 34-year-old female with a history of intravenous drug use and recent suicidal ideation was reported missing by her family in late winter. She was missing for a total of seven days before she was located by the police. During this time the outdoor temperature ranged from -10.4 to +1.2°C. She was found outdoors in a remote, wooded area and was transported by air to a tertiary care center.

Upon the paramedics arrival to the scene the patient was unresponsive with a Glasgow Coma Scale (GCS) of 4. Her pupils were 7 mm and non-responsive. She was severely hypothermic, with a rectal temperature of approximately 23 °C. She had a heart rate of 30 beats per minute, respiratory rate of 6 breaths per minute, and an unmeasurable blood pressure and pulse oxygen saturation. Peripheral pulses were not palpable in her limbs. Her extremities were diffusely cyanotic and had delayed capillary refill. Her legs were edematous and blistered bilaterally. Her mouth opening was 1 cm, and she bit down at any attempts to introduce an oral airway. Paramedics applied warming packs to her axillae and groin, obtained intravenous (IV) access and initiated fluid resuscitation with 1000 mL normal saline in an 18-gauge peripheral IV line at 75 mL/hr.

She was transported by rotary wing air ambulance from the scene to a tertiary care Level 1 trauma center. On presentation to the emergency department (ED), she was

identified to have a GCS of 3, with a heart rate of 40 beats per minute, respiratory rate of 8 breaths per minute, an unmeasurable blood pressure and pulse oxygen saturation, and a rectal temperature of 19.8 °C. Her cardiac rhythm showed sinus bradycardia. The patient's airway was secured by rapid sequence intubation with 2 mg midazolam, and 30 mg rocuronium. A femoral central venous catheter was inserted, and she continued to receive IV fluids in the form of two liters of Lactated Ringer's warmed to 41 °C through a femoral 3-lumen central line. A forced air rewarming unit was also applied. The patient was not observed to have any episodes of cardiac dysrhythmia during her time in the ED.

Rather than initiating invasive rewarming in the ED, in consultation with the cardiac surgeon a decision was made to transfer the patient directly to the operating room (OR) for CPB for the purpose of rewarming. The patient was transferred to the OR 40 minutes after presenting to the ED. At the time of transfer, the patient's rectal temperature was 20.2 °C. In the OR, the patient had a junctional escape rhythm of 50 beats per minute. Because of severe stocking-like erythema and edema in her lower extremities, IV fluid bags were placed under her feet to prevent ulcer development during the operation. She underwent a median sternotomy, revealing a dilated heart with a soft ascending aorta. She was cannulated with a two-stage venous cannula via atrial appendage, infused with heparin, and rewarmed at a rate of 12 °C per hour. The total OR time was 3 hours and 45 minutes, with 66 minutes spent on CPB. Upon transfer from the OR the patient had been rewarmed to a temperature of 36.9 °C as seen in Image. The patient was noted to be coagulopathic secondary to hypothermia and hemodilution, and therefore was administered protamine, fresh frozen plasma, platelets, and recombinant factor VIIa to reduce postoperative bleeding.

The patient was subsequently transferred to the intensive care unit. On post-operative day 5 she developed *Klebsiella oxytoca* and *Enterococcus fecalis* bacteremia and sepsis, attributed to severe skin and soft tissue infection of her bilateral lower extremities. This was initially treated with Piperacillin and Tazobactam. The patient was seen by plastic surgery for wound care. Her blisters and necrotic tissue were debrided and sterile moist dressing changes were performed daily. The patient was initially extubated and then re-intubated on post-operative day 7 because of increased oxygen requirements. Evidence of necrosis was noted in her toes on post-operative day 7. At this point her antibiotics were changed to imipenem and vancomycin. The patient was successfully extubated on post-operative day 13.

The consultant vascular surgeon recommended staged amputations of both lower extremities. Unfortunately, the patient was non-compliant with IV antibiotics and did not consent to surgery. The patient was assessed by psychiatry and found to be emotionally distant with a depressed mood but with no evidence of psychosis or delirium. She was deemed to be competent in her decisions. She was also offered addiction counselling but refused. Although the patient ultimately accepted oral antibiotic therapy, the condition of her lower extremities continued to worsen, and she eventually developed frank necrosis and dry gangrene. The patient expressed interest in hyperbaric oxygen treatment, and on post-operative day 39 she was transferred to another tertiary care center for foot-preserving therapy. Unfortunately, conservative measures and hyperbaric treatment were unsuccessful and

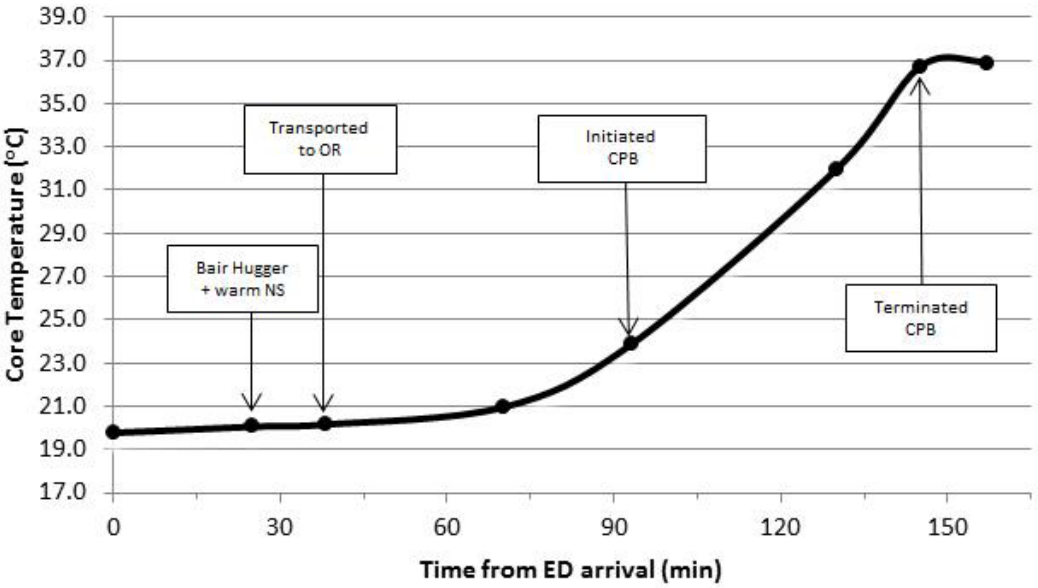

Image. Patient's core body temperature measurements in relation to time from arrival to the emergency department. Major intervention points have been outlined. NS=normal saline, CPB=cardiopulmonary bypass.
NS, normal saline; *CPB*, cardiopulmonary bypass, *OR*, operating room, *ED*, emergency department

Table. Blood sample measurements in relation to time of presentation to the ED and core body temperature.

Lab Parameter	38min 20.2°C	70min 21.0°C	Begin CPB ↓ 93min 23.9°C	130min 32.0°C	145min 36.7°C	End CPB ↓ 157min 36.9°C	In ICU ↓ 285min -	660min -
K^+ (mmol/L)	4.0	3.0	3.0	3.1	4.1	4.1	4.7	4.9
Na^+(mmol/L)	140	141	138	142	141	143	140	142
pH	7.08	7.16	7.12	7.32	7.36	7.39	7.42	7.43
pCO_2 (kPa)	59	35	-	29.8	28	29.9	33	35
pO_2 (kPa)	60	99	-	275	409	409	251	136
HCO_3 (mmol/L)	17	12	13	15.1	15	17.8	21	22
Glucose (mmol/L)	7.5	6.2	6.5	6.5	5.2	5.2	3.1	6.3

ED, emergency department; *CPB*, cardiopulmonary bypass; *ICU*, intensive care unit.

she ultimately consented to bilateral below-knee amputations, which took place 84 days after her initial presentation. Despite a total of up to seven days of severe accidental hypothermia, the treatments outlined above allowed the patient to make a complete neurologic recovery.

DISCUSSION

The patient in this case presented with a rectal temperature of 19.8 °C, meeting criteria for severe hypothermia. The risk of cardiac arrest is greatly increased with temperatures below 28 °C.[3] This justified the use of invasive central rewarming interventions, which are reserved for the most severe cases of hypothermia. The most rigorous central rewarming strategy is CPB, which uses extra-corporeal membrane oxygenation (ECMO) to permit oxygenation, filtration, or in this case, rapid warming of blood. ECMO may be complicated by hemorrhage (it requires systemic anticoagulation) renal failure, neurologic impairment, and pulmonary edema.[4-6] This case is unique since the patient survived without any of the aforementioned severe complications and made a complete neurologic recovery.

Most cases that involve ECMO for rewarming have used femoral-femoral-bypass, and report mortality rates ranging from 16-87% depending on the cause of hypothermia.[4,5,7,8,9] The patient in this case received an open bypass by sternotomy. Sternotomy was used to access the central vessels rather than peripheral cannulation because the patient's femoral vessels were small and cannulating them may have caused damage and sub-optimal flow rates. Another benefit of this approach was that it could allow for cardiac massage, which would lead to higher flow rates thereby allowing faster rewarming.[8]

There is lack of agreement in the literature regarding the appropriate rate approach to rewarming a severely hypothermic patient. A recent review by Brown et al. (2012) advised clinicians to consider multiple factors when selecting the rate, such as accessibility to an appropriate facility, local expertise, and patient characteristics. While CPB is certainly the fastest rewarming method at approximately 9 °C per hour, slower rates are often preferred.[2] This reluctance may be driven by the concern for the "afterdrop" effect, which describes paradoxical exacerbation of the core hypothermia secondary to peripheral vasodilation and return of cold peripheral blood to the body core.[1] Controlled hypothermia experiments have demonstrated that the afterdrop effect is overestimated and clinically insignificant.[11-13] The patient described in this case was rewarmed at a very rapid rate of 12 °C per hour, which is 8 °C per hour faster than is typically seen in venous-venous ECMO, and 3 °C per hour faster than in standard CPB.[2] The patient was rewarmed completely up to 36.2 °C with no afterdrop effect, and no other complications. Overall, there was no evidence suggesting that a faster rewarming rate was detrimental to the patient in this case.

Prehospital care of the severely hypothermic patient is critical, and the team treated the patient in this case expertly. Traditional literature advocates the avoidance of moving severely hypothermic patients brusquely, out of concern for their predilection to dysrhythmias, particularly ventricular fibrillation.[10,14] These concerns are poorly supported,[1,15] and it is our opinion that the prehospital team was correct to secure the patient in the transport vehicle and transport her to hospital as quickly as possible. The prehospital team was only at the site where the patient was found for five minutes before rapidly transporting her. Prompt evaluation and rapid transport by prehospital staff is what is suggested in the current literature[2,16].

Another consideration when rewarming hypothermic patients is the endpoint of therapy. There is discussion regarding whether a 24-hour course of "therapeutic hypothermia" at 32-34°C is indicated in this population; however, further research is needed to provide supporting evidence.[2] It seems appropriate to us to implement some form of targeted temperature management / therapeutic hypothermia only if there is suspicion of a concomitant anoxic insult.

Overall, effective prehospital care followed by rapid open sternotomy to allow CPB led to the survival and complete neurologic recovery of a 34-year-old woman found with a

core temperature of 19.8 °C. While CPB is an effective and well-documented treatment for patients with hypothermia and cardiovascular collapse, we present a case of successful treatment of prolonged severe hypothermia with CPB in the absence of cardiovascular collapse. Many people die from accidental hypothermia every year and even more are exposed. These patients are not uniformly treated, and there is a paucity of guidelines and standards governing their treatment. This remarkable case demonstrates a viable option for treatment of such patients, and can be used to guide the development of future protocols.

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Cervical Ectopic Pregnancy in a 23 Year Old with Uterus Didelphys

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Ectopic pregnancy remains an important diagnosis for the emergency physician to recognize, accounting for up to 2% of all pregnancies and associated with significant morbidity and mortality. Ectopic pregnancies can implant in various sites outside of the uterus, one of the rarest of which is in the cervix. Cervical ectopics account for less than 1% of ectopic pregnancies, but are associated with higher rates of significant bleeding than others.[1-2] Uterine anomalies are a predisposing factor for ectopic pregnancies. This case highlights the management of a cervical ectopic pregnancy in a 23 year old with a history of uterine didelphys. [Clin Pract Cases Emerg Med. 2017;1(1):37–39.]

INTRODUCTION

Ectopic pregnancy is an important diagnosis to consider in a fertile female who presents to the emergency department with vaginal bleeding and/or abdominal pain. It is defined as the implantation of the zygote outside of the uterine cavity. Ectopic pregnancies are associated with various complications that can lead to significant morbidity and mortality if not promptly and appropriately managed[1]. Hemorrhage from ectopic pregnancy remains the leading cause of mortality in the first trimester.

Ectopic pregnancies can occur in multiple places. 98% of ectopic gestations occur in the fallopian tubes. Cervical pregnancy is rare, accounting for <1% of ectopic pregnancies[2]. Due to a paucity of information on cervical ectopic pregnancies, the most effective treatment has not been clearly defined.

Generally, there are two mainstays of treatment of ectopic pregnancies; surgical and medical. Surgical therapy involves manually extracting an ectopic pregnancy and any other affected anatomic structures as clinically indicated. Medical therapy involves the administration of an abortive medication. Currently, the most common agent used is methotrexate, a folic acid antagonist that inhibits DNA synthesis in actively dividing cells[3]. Several studies have demonstrated that methotrexate was as effective as laparoscopic treatment of ectopic pregnancies when used appropriately[4-6]. (Table 1)

Table 1. Favorable criteria for medical management of ectopic pregnancy.

Criteria
Stable hemodynamics
Compliance
Post-treatment follow-up
Human chorionic gonadotropin concentration ≤5000mIU/mL
No fetal cardiac activity
Ectopic mass size ≤3cm

Risk factors for ectopic pregnancies include anatomic anomalies, prior ectopic pregnancies, in utero diethylstilbestrol exposure, implantation in the presence of an intrauterine device, smoking, previous genital infections, and in vitro fertilization. Uterine anomalies arise from abnormal embryological development of the Mullerian ducts. Of the Mullerian duct abnormalities, a didelphic uterus is one of the rarest, making up just 8.3% of all Mullerian duct abnormalities[7]. A didelphic uterus results from incomplete failure of the Mullerian ducts to fuse resulting in two separate uterine cavities, two cervices, and often vaginal abnormalities such as double vagina or a longitudinal vaginal septum[14]. It can be associated with Wolffian duct abnormalities resulting in kidney abnormalities as well.

We report a case of a 23 year old female who presented with vaginal bleeding and was found to have a cervical ectopic pregnancy and a didelphic uterus.

CASE REPORT

A 23 year old female G1P0, with a history of hypothyroidism and polycystic kidney disease presented to the ED with 1 week of pelvic pain and 1 day of vaginal bleeding. She described the pelvic pain as crampy and intermittent with no specific relieving or exacerbating features. The day prior to presentation, she noticed heavy vaginal bleeding and described the passage of clots and tissue.

Upon presentation, she was hemodynamically normal. Her physical exam was notable for mild lower abdominal tenderness to palpation. Her pelvic exam revealed normal appearing external female genitalia, a normal appearing cervix in the midline, and a smaller cervix to the left, without obvious products of conception. There was evidence of a small partial longitudinal septation between the two cervices. Bimanual exam was unremarkable. Laboratory evaluation was significant for a serum beta hCG of 6,045 mIU/mL, B positive blood type, a hemoglobin of 13.7 g/dL with a hematocrit of 41.5%. Transvaginal ultrasound was notable for two uteri, two cervices and a gestational sac without yolk sac or fetal pole present in the left endocervical canal (Image 1, Image 2).

The patient was diagnosed with a cervical ectopic pregnancy, and OB/GYN was consulted and evaluated the patient at bedside. The patent was discharged home with 48 hour follow up with OB/GYN. Repeat ultrasound in clinic showed persistence of gestational sac in left endocervical canal as noted in prior imaging study. Methotrexate therapy was initiated, and the patient received 81mg IM x 1 in clinic 2 days after she was seen in the Emergency Department. She had weekly follow-up with repeat B-HCG levels (Table 2) following the methotrexate dose. By week 4, her β-hCG levels and repeat ultrasound revealed resolution of the cervical ectopic. The patient was counseled on birth control and to seek early OB care if pregnant again in the future.

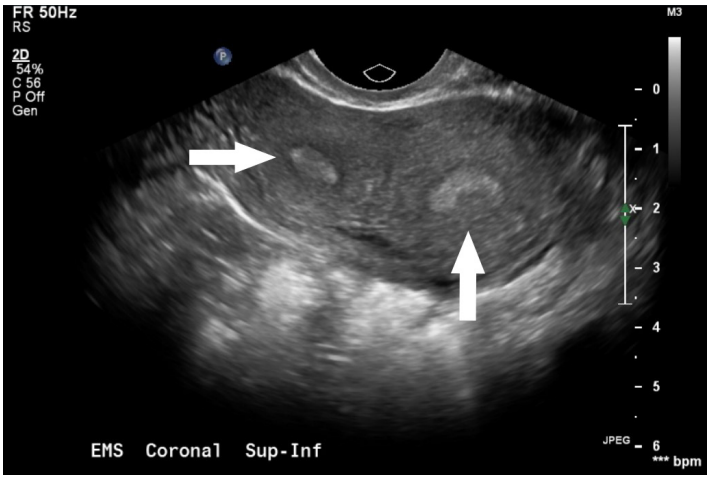

Image 1. Transvaginal ultrasound demonstrating two uterine cavities (arrows).

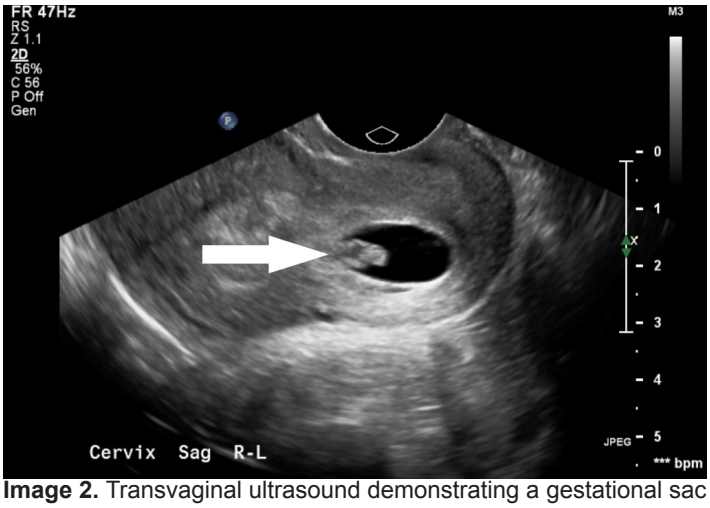

Image 2. Transvaginal ultrasound demonstrating a gestational sac (arrow) in the left endocervical canal.

Table 2. Human chorionic gonadotropin concentration (hCG) of the patient from November 7 to December 7.

Date(2015)	hCG concentration mIU/mL
November 7	6045
November 9	2789
November 13	675.7
November 16	265.9
November 23	52.9
November 30	8.9
December 7	3.1

DISCUSSION

To our knowledge, this is the first reported case of a cervical ectopic in a patient with a didelphic uterus in the emergency medicine literature. Ectopic pregnancies account for 6-16% of all causes of first trimester bleeding[3]. Hemorrhage resulting from ectopic pregnancy is the leading cause of maternal death in the first trimester and has been reported to be up to 10% of all pregnancy-related deaths[8]. Most ectopic pregnancies occur in the fallopian tube and <1% occur in the cervix. Cervical ectopics are important to recognize in the Emergency Department due to the increased risk of life-threatening bleeding due to erosion into the cervical blood vessels. Predisposing factors for cervical ectopic pregnancies include prior dilation and curettage, prior caesarean section, and in vitro fertilization. While this patient presented in stable condition, some ectopic pregnancies can present with severe vaginal bleeding.

Emergent therapies beyond rapid consultation with a gynecologic surgeon include foley balloon tamponade of the cervix and vaginal packing.

There are many risk factors that predispose individuals to developing ectopic pregnancies. It is important for the emergency physician to be aware of such risk factors, including rare conditions such as a didelphic uterus. Emergency providers should be cognizant of didelphic uterus due to the expected abnormalities on physical exam, as well as the importance to fully visualize all of the relevant anatomy on ultrasound.

The optimal management of cervical ectopic pregnancy in a patient with a didelphic uterus remains unclear. Due the paucity of information present on cervical ectopic pregnancies in patients with a didelphic uterus, it is unknown whether or not medical or surgical management is favored[2]. The information presented on management of cervical ectopic is limited to isolated case reports and small retrospective studies[9-12]. Many of these reports discuss the use of medical therapy of cervical ectopic with a secondary goal of preserving the uterus for potential future pregnancies. However, none of these reports discuss the management of cervical ectopic pregnancy in a patient with a history of didelphic uterus and the fertility of individuals who have this anatomy is not well defined[13]. This case demonstrates the successful management of a rare presentation of ectopic pregnancies with single dose methotrexate. In a hemodynamically normal patient with close follow-up and mild to moderate bleeding, it may be appropriate to discharge these patients from the ED with strict return precautions. As with most ectopic pregnancies, we recommend expert consultation with OB/GYN.

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Methadone-induced Torsades de Pointes Masquerading as Seizures

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The authors herein present the case of a 53-year-old female who was being treated as an outpatient for seizure disorder but was also receiving high-dose methadone therapy. She presented to the emergency department (ED) for what appeared to be a seizure and was found to have a prolonged QT interval, as well as runs of paroxysmal polymorphic ventricular tachycardia with seizure-like activity occurring during the arrhythmia. The markedly prolonged QT interval corrected after treatment with intravenous magnesium; subsequent electroencephalogram, neurology and cardiology consultations confirmed the cause of the recurrent seizure-like episodes to be secondary to the cardiotoxic effects of methadone. [Clin Pract Cases Emerg Med. 2017;1(1):40–43.]

INTRODUCTION

QT interval prolongation and associated torsades de pointes (TdP) are well-documented side effects of methadone. Moreover, case reports illustrating the development of TdP in patients using methadone appear to be associated with higher doses of the medication.[1] TdP is usually a self-remitting non-perfusing arrhythmia commonly manifesting as syncope or seizures. This case highlights the need to consider arrhythmias in the differential of a patient with seizures and/or presumed seizure disorder and emphasizes the importance of obtaining an electrocardiogram (EKG) in such patients.

CASE REPORT

A 53-year-old Caucasian female presented to the emergency department (ED) after having what appeared to be a single seizure just prior to arrival, witnessed by her husband. He described generalized shaking lasting approximately 10 seconds with an associated brief period of loss of consciousness but no reported gaze defect, bowel or bladder incontinence, apnea or tongue biting. There was a 10-15 minute period of decreased responsiveness and confusion that occurred after the event. The patient's husband reported she was evaluated at another ED two weeks prior for a similar episode. During that visit her labs and computed tomography (CT) of the head were negative. The patient refused admission at that time because she was concerned that she would not receive her methadone. Subsequently, she was given a prescription for levetiracetam and followed up with a neurologist the next day.

On the day of her presentation to our ED she was on her way to a scheduled EEG and magnetic resonance imaging of her brain. The husband reported that the patient had been having one to two seizures daily for the preceding two weeks, despite taking the prescribed dose of levetiracetam. The patient stated that she did not recall having the seizures; however, she described feeling palpitations and shortness of breath just prior to developing these episodes. There was no prior history of seizure disorder or recent head trauma. Her past medical history was significant for anxiety, for which she took alprazolam 0.5 mg twice daily, and heroin dependence for which she took methadone 140 mg daily. Emergency medical services reported her blood sugar to be 125 mg/dl and she appeared post-ictal with mentation that gradually improved during transport. On arrival to the ED, the patient was awake, well developed and well groomed, but was slow to respond. Her vital signs were blood pressure 129/81 mmHg, pulse 62 beats per minute (bpm), respiratory rate of 17 breaths per minute, temperature of 97.3 degrees Fahrenheit, and pulse

oximetry of 100% on room air. There were no obvious signs of head trauma and pupils were round, 3 mm bilaterally and reactive to light with extra-ocular muscles intact. Cardiac, lung and abdominal exam were unremarkable. The patient was alert and oriented to person, place, time and situation. Her Glasgow Coma Scale was 15 and she had no focal neurological deficits.

On arrival to the ED the patient was placed on a cardiac monitor and an EKG was obtained (Image 1). The EKG showed sinus bradycardia at a rate of 59 bpm with normal axis but the QT was prolonged at a rate of 622 msec. The patient was immediately given magnesium 2 grams intravenous and laboratory exams were obtained, which returned unremarkable. While in the ED the patient was noted to have multiple premature ventricular complexes on the telemetry monitor, which developed into two separate short runs of non-sustained paroxysmal polymorphic ventricular tachycardia (Image 2). During the first of these episodes the patient reported feeling palpitations and shortness of breath. During the second, longer event, the patient was witnessed to have an abbreviated episode of generalized tremor, which the husband reported to be similar to her prior occurrences of seizures. She also appeared post-ictal after this episode for approximately five minutes. Her blood pressure remained stable throughout the entire course. In addition, the patient received additional intravenous infusions of magnesium during and after these episodes. A CT of the head was obtained in the ED and was unremarkable for any acute pathology. A repeat EKG was obtained prior to patient transfer to the cardiac care unit, and after the magnesium infusions, which showed shortening of the QT length to 363 msec.

During her hospitalization neurology and cardiology consultations were obtained. An electroencephalogram was unremarkable for evidence of seizure activity and the cardiologist and neurologist collectively agreed that the prolonged QT and development of non-sustained polymorphic ventricular tachycardia were likely secondary to the high dose of methadone and the cause of the perceived seizure disorder. The patient's QT interval remained within normal limits after receiving the doses of magnesium in the ED and throughout the remainder of her admission to the hospital. There was no recurrent seizure-like activity or arrhythmias during her hospitalization. She declined the placement of a defibrillator, which was recommended by cardiology since she refused to decrease her methadone dose. Both the physician who prescribed the patient's methadone and her primary care physician were notified about her hospital course and work-up. Despite her reluctance, the patient was tapered to a lower

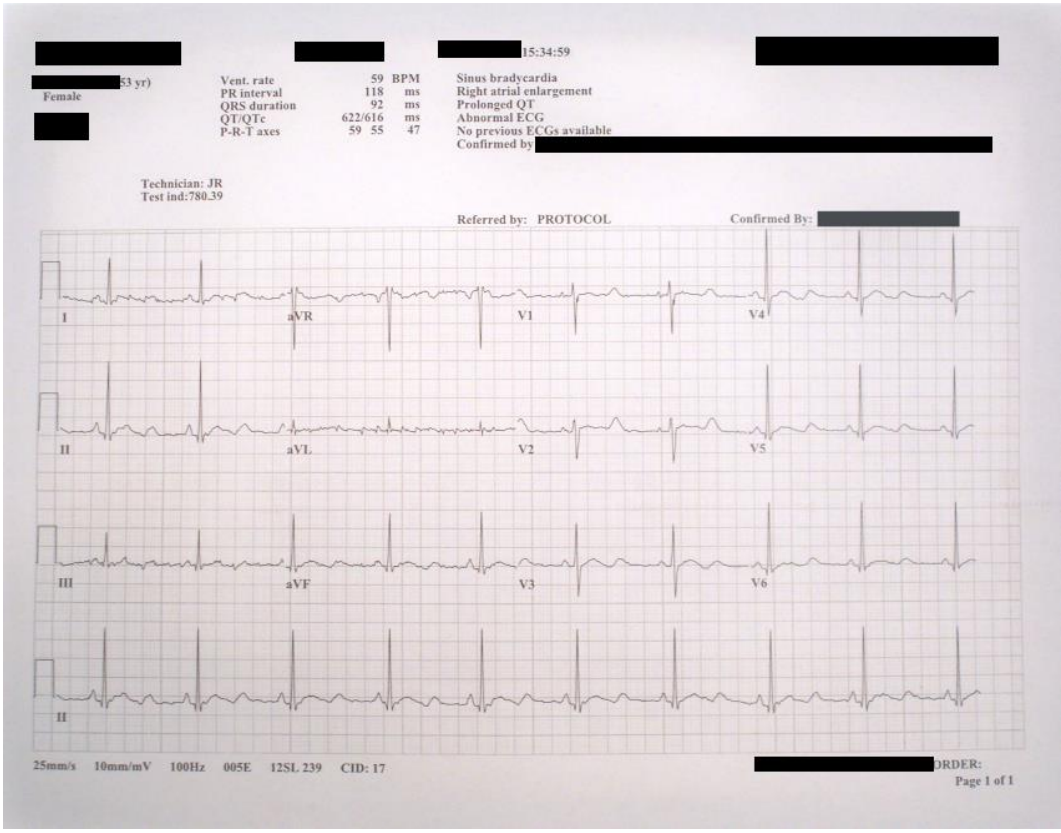

Image 1. Initial electrocardiogram on patient arrival to the emergency department.

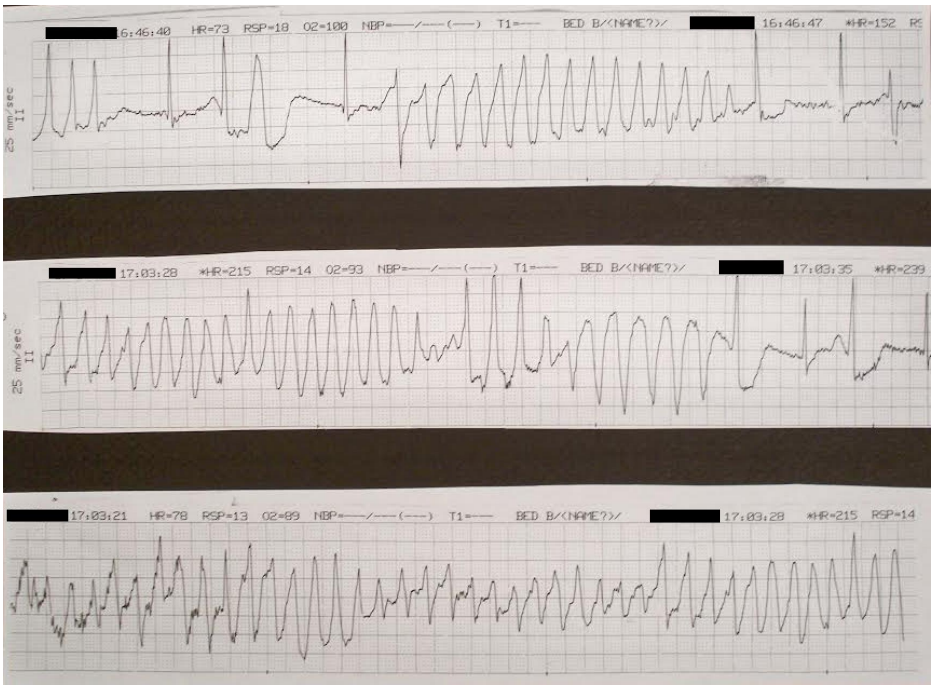

Image 2. Rhythm strip demonstrating runs of Torsades de Pointes.

dose of methadone and was subsequently followed closely by her primary care physician for monitoring of her EKG. In addition, she was prescribed magnesium oxide 400 mg tablets twice daily prior to her discharge.

DISCUSSION

Methadone is a long-acting synthetic opioid that can be used for pain control; however, its main use is as a maintenance program in patients with a history of heroin abuse.[2] It has several known side effects, most importantly respiratory depression and cardiac toxicity. Specifically, methadone has been shown to increase QT intervals and is associated with the development of TdP. The development of QT prolongation by methadone is secondary to blockade of the delayed rectifier potassium current through the cardiac human ether-a-go-go related gene (hERG) channels.[3] This occurs in a dose-dependent fashion putting those on chronic therapy with higher doses to be at risk for QT prolongation.[4] The incidence of some degree of QT prolongation has been reported in more than 80% of patients on methadone maintenance therapy. More profound QT prolongation (>500 msec) in this population, however, has been reported in the literature to have an incidence of 2.4% to 16.6%.[5,6] The exact incidence of methadone-induced TdP is unknown; however, separate studies report an incidence of between 0.3% to 3.6%.[7,8] TdP is a specific form of polymorphic ventricular tachycardia that is commonly associated with patients with prolonged QT interval. Patients with TdP may present with sudden cardiac arrest, but more typically present with recurrent episodes of dizziness, palpitations, syncope or what may be perceived as seizure.[9-11] The reporting of seizure occurring during an episode of TdP has been reported in the medical literature.[12-15] Our patient was presumed to have a recently diagnosed seizure disorder and was started on antiepileptic medication; this differs from previously reported cases in which the patients were not being treated for seizure disorder as outpatients. The witnessing of what appeared to be a seizure occurring during an episode of TdP in our patient, as well as the negative work-up for seizures, solidifies the diagnosis of TdP-induced central nervous system hypoperfusion appearing to be a seizure. Although our patient was also on alprazolam, the potential for alprazolam withdrawal as the etiology of her seizures is unlikely as she was reportedly compliant with her medications. In addition, apart from alprazolam and levetiracetam, which may have mildly contributed to the patient's QT interval, she was not taking any other medications that could have prolonged the QT interval.

CONCLUSION

The authors believe the patient's manifestation of seizure episodes was secondary to the cardiotoxic effects of her high-dose methadone treatment. After receiving intravenous magnesium in the ED, with subsequent improvement in the QT interval and resolution of further episodes of TdP, the patient had no further episodes of seizure activity. More likely there were

no epileptic seizures; instead, epileptiform motor activity was secondary to cerebral hypoperfusion due to runs of paroxysmal polymorphic ventricular tachycardia. This case serves as an important reminder for why EKGs are vital diagnostic tools for a patient presenting with seizures, especially for those patients taking methadone or other drugs that are associated with QT interval prolongation. Healthcare practitioners should consider obtaining an EKG in patients presenting with seizures and should inquire about the use of methadone and other drugs that can predispose a patient to arrhythmias.

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Valentino's Syndrome: A Life-Threatening Mimic of Acute Appendicitis

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Perforated ulcers are a rare cause of abdominal pain, and may not be considered when pain is localized to the right lower quadrant (RLQ). This case highlights an unusual presentation of a perforated duodenal ulcer that presented with RLQ pain, which has been described as Valentino's syndrome. Valentino's syndrome occurs when gastric or duodenal fluids collect in the right paracolic gutter causing focal peritonitis and RLQ pain. This case highlights that perforated ulcers, while an uncommon cause of RLQ pain, must remain on the differential of any patient that has an abdominal examination consistent with peritonitis. [Clin Pract Cases Emerg Med. 2017;1(1):44–46.]

INTRODUCTION

Approximately six million people in the United States have peptic ulcer disease (PUD) with an annual direct cost estimate of $3.1 billion.[1] *Helicobacter pylori* infection and nonsteroidal anti-inflammatory drug (NSAID) use are the two main causes. With improved understanding of the pathogenesis of PUD and treatment with antibiotics, proton pump inhibitors (PPIs), avoidance of NSAIDs and advanced endoscopy techniques, the incidence of complications from PUD requiring surgical intervention has decreased to approximately 11%.[2] While a rare event, ulcer perforation is associated with high mortality at 10.6%, and emergency physicians (EP) must remain vigilant.[2] This case highlights Valentino's syndrome, in which the patient presents with pain in the right lower quadrant (RLQ) of the abdomen due to perforation of a duodenal ulcer through the retroperitoneum.[3,4,5,6] To our knowledge, this is only the second reported case of Valentino's syndrome in the emergency medicine literature.

CASE REPORT

An 18-year-old woman presented to the ED with one-day history of RLQ abdominal pain. She described the pain as constant with an acute worsening approximately three hours prior to arrival. The pain was sharp, 10/10 in intensity, and associated with diaphoresis, nausea, vomiting, and anorexia. She reported taking ibuprofen for her symptoms, which provided little relief. She denied a history of dysuria, change in vaginal discharge, or being sexually active. She was seen by her primary care physician 12 days prior with similar but less severe pain. She was diagnosed with gastritis and prescribed ranitidine 150mg twice daily and ondansetron 4mg as needed for her symptoms. She was otherwise healthy, with no reported prior medical problems or surgeries. She was a senior in high school, lived with her parents and did not smoke, drink alcohol or use illicit substances.

On physical exam the patient's vital signs were temperature 98.0°F, pulse 85, blood pressure 128/72, respiratory rate 18, and pulse oximetry 98% on room air. She was well nourished but in obvious distress from pain. Abdominal examination revealed tenderness over McBurney's point with focal peritonitis. Pelvic exam was unremarkable, with normal discharge and without cervical motion tenderness or adnexal masses or tenderness. Rectal examination was performed, which demonstrated brown Guaiac negative stool.

Laboratory data were remarkable for a negative urine HCG. Urinalysis showed small leukocyte esterase, nitrite negative and without hematuria or pyuria on microscopic analysis. Her white blood cell count was 6,200 mcl, otherwise normal complete blood count, electrolytes and creatinine. Serum lactate was 0.96 mEq/L. Liver function tests showed an elevated LDH of 519 U/L, normal bilirubin and transaminases. Lipase was elevated at 87 U/L.

The time course of symptoms and the degree of distress initially led the EP to consider ovarian torsion, but with

the tenderness localizing to McBurney's point and normal pelvic examination, a computed tomography (CT) of the abdomen pelvis was ordered. The study was protocoled for appendicitis (intravenous contrast only). This study revealed pneumoperitoneum centered within the greater sac of the upper anterior abdomen and free fluid in the pelvis (Images 1 and 2). This was favored to be secondary to a perforated duodenal ulcer and general surgery was emergently consulted.

The patient was admitted to the general surgery service and underwent an exploratory laparoscopy. She was found to have a small, perforated ulcer in the first portion of her duodenum, which was repaired with a tongue of omentum, known as a Graham patch.[7] Her postoperative course was uneventful. Serologic testing for *H. pylori* antibody was significantly elevated at 2.8, U/ml, indicating an active infection, and her gastrin level was normal. She was treated for *H. pylori* with amoxicillin/clarithromycin/bismuth and was discharged on postoperative day 3. Four weeks later, upon completion of triple therapy, upper endoscopy demonstrated chronic gastritis and biopsies were negative for *H. pylori*.

DISCUSSION

Valentino's syndrome occurs when gastric or duodenal fluid collects in the right paracolic gutter causing focal peritonitis and RLQ pain.[3,4,5,6] The syndrome is named after the 1920s silent film star Rudolph Valentino. In 1926, Valentino collapsed in a New York City hotel and underwent surgery for presumed appendicitis at New York Polytechnic Hospital. At the time he was found to have a perforated ulcer. Postoperatively, he developed peritonitis, multiple organ system dysfunction and died several days later.[8] His case gained significant notoriety due to his fame at the time, and has since become a cautionary tale to medical students and residents.

Gastric and duodenal ulcers are often collectively referred to as PUD because of the similarity in their pathogenesis and treatment. *H. pylori* and NSAIDs contribute the most to PUD. Complications from PUD include hemorrhage, obstruction, cancer, and perforation. A better understanding of the risk factors for PUD has led to a significant decrease in complications of the disease. Total admissions for PUD have decreased by almost 30% since the 1990s. The percentage of patients who require emergent surgery for complicated disease has decreased, to approximately 11%. Perforation has the highest mortality rate of any complication of ulcer disease, approaching 15%. Despite a decrease in reported ulcer-related mortality, from 3.9% in 1993 to 2.7% in 2006, over 4,000 estimated deaths are caused by PUD each year.[2]

Perforation of an anterior duodenal ulcer allows for free communication of duodenal and gastric contents into the peritoneal cavity. These contents will collect in dependent portions of the peritoneum, which is often the RLQ.[3] If the patient seeks medical attention early in the course of the disease, he or she may have poorly localized pain. Localized pain to the RLQ can mimic acute appendicitis so closely that surgical exploration without imaging has lead to the diagnosis being made intra-operatively.[3] As time passes, this can progress to focal tenderness, as in this case, or to generalized peritonitis. Initial imaging other than CT may demonstrate free fluid around a normal appendix on ultrasound and free air around the kidney, or "veiled kidney sign" on abdominal radiographs.[6] This patient was hemodynamically stable with focal peritonitis consistent with a surgical abdomen with the diagnosis made on CT imaging.

Definitive treatment for a perforated duodenal ulcer is surgical. This patient underwent a laparoscopic Graham

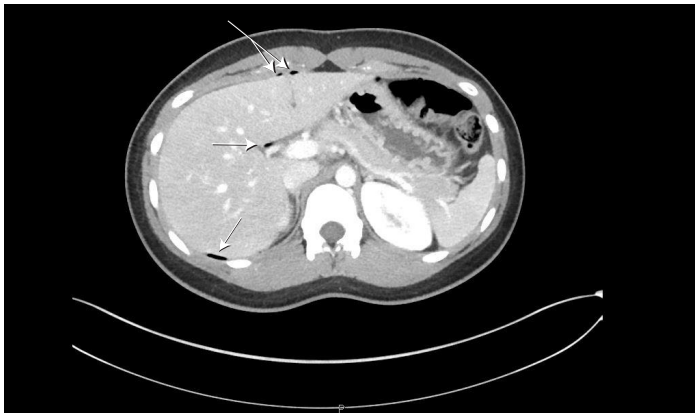

Image 1. Axial CT image demonstrating free air (arrows) in superior abdomen.

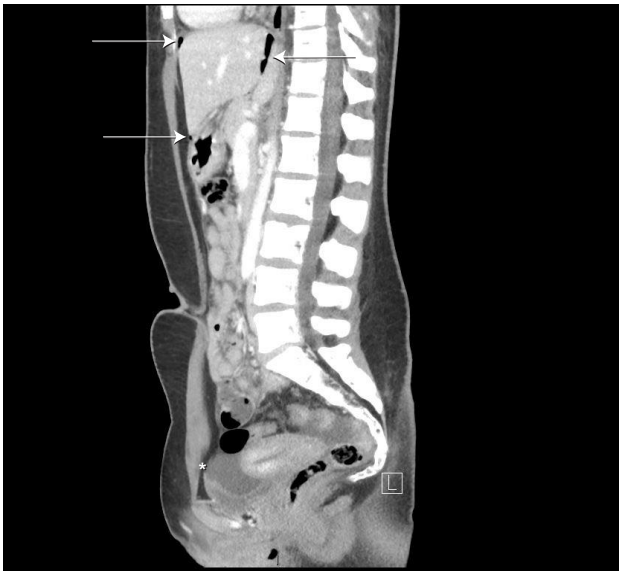

Image 2. Sagittal CT image demonstrating free air (arrows) and free fluid (asterisk) in abdomen.

patch repair and had an uneventful postoperative course. According to primary care notes, she reported several weeks of abdominal pain, likely a result of gastritis/PUD, presumably from her *H. pylori* infection. Treatment with NSAIDs may have exacerbated her disease, leading to her complication of perforation. Given her age, Zollinger-Ellison syndrome – a gastrin-secreting tumor, most commonly found in the exocrine pancreas – was also on the differential. However, a serum gastrin level was obtained which was normal, effectively ruling out this disease.

CONCLUSION

While rare, especially in young people, perforated gastric and duodenal ulcers have a high morbidity and mortality. Air and liquid from perforated viscous can track to various locations in the abdomen. Thus, in any patient with a peritoneal exam, regardless of the location of this pain, perforated ulcer should remain on the differential.

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Remote South American Snakebite with Extensive Myonecrosis

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This report describes a patient envenomated by a *Bothrops atrox*, common fer-de-lance viper, in the remote rainforest of eastern Ecuador and without access to definitive care for seven days. Antivenom was not administered; by the time of presentation to a hospital, he had suffered myonecrosis of his lower leg, which was treated with debridement and eventual skin graft. The ramifications of this long evacuation demonstrate the need for more accessible health services and educational outreach. [Clin Pract Cases Emerg Med. 2017;1(1):47–49.]

INTRODUCTION

The estimated 400,000 snakebites that occur annually worldwide are a serious problem for the regions they affect, primarily in nations with lower income and resources.[1] Over 1,000 snakebites occur each year in Ecuador, with the vast majority inflicted by various pit viper species of the *Bothrops* genus.[2] Furthermore, *B. asper* is responsible for the majority of ophidic morbidity and mortality throughout Latin America. Studies have demonstrated that incidence and death rates (10 and 0.05 per 100,000 persons per year, respectively) in Ecuador are consistent with neighboring rural Brazil and Colombia.[3,4] In a study of 187 bites treated with antivenom in the Amazon region of Ecuador, two cases resulted in death; common complications included cellulitis (16%), renal failure (8%), abscess (6%), compartment syndrome (6%), and local necrosis (5%).[2] Multiple studies show that polyvalent antivenoms from a variety of southern Central or South America countries have similar efficacies at reducing death as well as the hemotoxic and myotoxic effects of vipers from other regions.[1,3,5] This has allowed countries such as Costa Rica to produce the vast majority of antivenoms used in Latin America, so that other countries need not invest considerable resources into production. Despite this, antivenoms are often unavailable in remote and rural regions due to the need for refrigeration and restrictive cost ($300 U.S. per vial). This unfortunately results in delayed treatment and increased complications.[6]

Patient Presentation

A 36-year-old Ecuadorian male was transferred from a rural health center to a district-level hospital with worsening right lower leg pain, edema, and skin discoloration following a snakebite in a remote area of the Amazon seven days prior. He stated that he was walking in the jungle barefoot when he sustained an unprovoked bite on his right ankle. He beheaded the snake and walked to a neighboring shaman who provided local herbs and prayer for one day. He required several days of remote travel by foot and canoe to arrive at a community where air evacuation could be arranged for the remainder of the 150 miles to the district hospital. He identified the snake as an "equis," the common name in the area for the *B. atrox*. His associated symptoms included gingival bleeding, hematemesis, abdominal pain, oliguria, and shortness of breath. He denied fever, chest pain, or neurological changes.

Triage vital signs were temperature 36.7 ºC, blood pressure 100/60 mm Hg, heart rate 104 beats/min, respiratory rate 20 breaths/min, and SpO2 95%. Physical exam revealed the patient in no acute distress. His lower leg was edematous with areas of hyperpigmentation and ulceration extending to the dorsal foot. Labs showed a 12-minute whole blood clotting time (WBCT, normal 80-160 seconds), creatinine of 3.25 (0.5–1.2 mg/dL), urea of 174 (7 to 20 mg/dL), and a normocytic anemia with a hemoglobin of 5.3 (13.5-17.5 g/dL) and hematocrit of 14.1(43-52%). Ultrasound of the leg showed

preserved blood flow and a fluctuant (4 cm x 1 cm) area in the anterior compartment; incision and drainage yielded 4mL of purulent fluid. Abdominal ultrasound revealed decreased renal corticomedullary differentiation suggestive of inflammation without obstruction.

The patient was diagnosed with a viper bite complicated by an abscess of the anterior tibial compartment and grade 3 renal insufficiency. The patient received two units of packed RBCs and was started on intravenous fluids, erythropoietin, iron, folic acid, enalapril, oxacillin, and acetaminophen. Subsequently, the patient was transitioned to clindamycin and started on enoxaparin for venous thrombosis prophylaxis. His necrotic tissue required staged debridements in the operating room, resulting in a large wound with exposed tendons; the wound was treated with daily bandage changes and sugar application for one month prior to successful skin grafting (Image 1). All care was provided by surgeons at the district level hospital in Macas with the exception of skin grafting in a tertiary care facility in Cuenca. We advised the patient to remain until full healing of the skin graft to reduce chance of infection in the remote jungle, but the patient wanted to return home to family and left against medical advice after two months of care.

DISCUSSION

This case emphasizes the sequela of delayed definitive care in snakebites, which occurs far too commonly in rural areas of Ecuador. The risk of dying or developing serious complications from a snakebite is significantly higher (relative risk = 2.5) in those who do not receive antivenom in the first two hours after a snakebite.[5] A polyvalent antivenom from Costa Rica was available for this patient once he reached the district-level hospital; however, given that the bite had occurred over one week prior to presentation, it was not administered. WBCT of greater than 20 minutes is often used to assess presence of viper bite; in this case WBCT of 12 minutes likely indicated decreased venom load and resolving coagulopathy. A study of the kinetics of *Bothrops* venom in rats showed an elimination half-life of 12 hours, with no circulating venom detected after seven days postenvenomation.[7] It is unlikely that this patient would have experienced any benefit from antivenom if there was no remaining venom to be neutralized. Furthermore, few data exist on antivenom treatment timelines, but the high rate of adverse reactions to antivenoms (15–25%) makes discretionary use prudent.[1]

Snakebite treatment protocol exists at the hospital in which the man was treated; however, given that the bite occurred over seven days prior to presentation, deviation from the protocol occurred and the patient was treated according to protocol and expert opinion. As a result, the patient was treated primarily using the indicated supportive treatment. Image 2 depicts the standard snakebite protocol used in Latin America that was developed in Costa Rica.[2,8]

Hemotoxic effects such as this patient's gingival bleeding and anemia are common and mediated by a variety of venom components including zinc-dependent metaloproteinases.[1,3,9] The extensive myonecrosis seen in this patient is thought to be largely due to toxic analogues of phospholipase A2 that result in phospholipid hydrolysis and compromise of the cell membrane. These toxic enzymes are the major target of the antibodies in antivenom, which neutralize and aid in their elimination.[1,2,3] Regrettably, necrosis following viper envenomation frequently results in deficient skeletal muscle regeneration secondary to loss of basement membrane and axons. This patient had minimal capability to dorsiflex and invert his foot when he left the hospital two months after his ophidian accident.[3]

Our patient was geographically isolated and spent days receiving shamanistic care. These are common barriers for those living in remote rainforest communities that prevent prompt definitive care of snakebites. Approximately half of those bitten in Latin American countries will be treated first

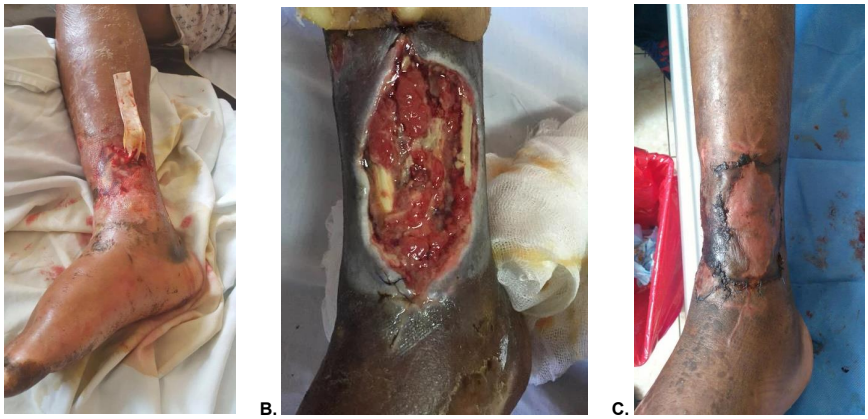

Image 1. Progressive treatment of extensive myonecrosis in snakebite victim. **A.** 10 days after bite with drain insert into abscess. **B.** Two weeks after bite and extensive debridement. **C.** Status post graft hospital day 50.

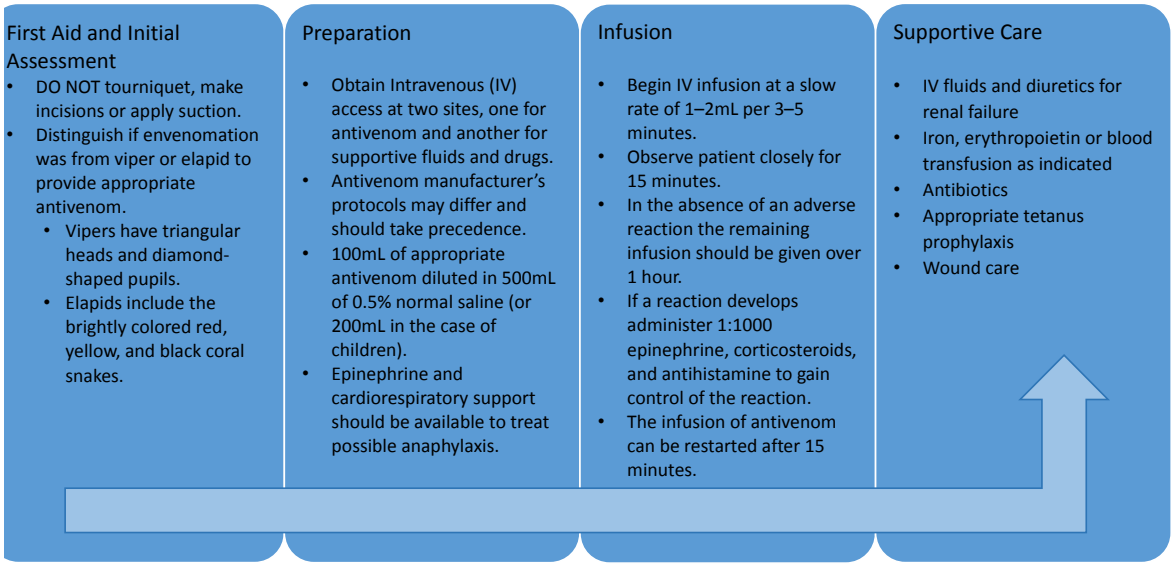

Image 2. Protocol for snakebite treatment. Adapted from Howes *et al.*, 2005[2] and De La Hoz *et al.*, 2008.[8]

with traditional methods such as plants, chemical products, physical methods, or prayer.[5] Unfortunately, these patients are isolated by long distances and geographical obstacles; traditional medicine is accessible and trusted, but only serves to delay antivenom administration. Efforts to educate traditional medicine practitioners and encourage rapid evacuation may serve to improve outcomes in remote regions. Despite Costa Rica's high incidence of snakebite (14 per 100,000 per year) the mortality rate is lower than in most similar nations (0.02 per 100,000 persons per year).[4] For 45 years the Instituto Clodomiro Picado in Costa Rica has had extensive educational outreach programs involving medical students and house staff to targeted high-risk communities using both Spanish and indigenous languages to improve knowledge of snakebite prevention and emergent treatment.[3] Existing diet, sexual, and maternal health educational outreach programs to rural Latin America may benefit from pilot programs that bring awareness to snakebite preventative measures and the ramifications of delayed antivenom treatment. The morbidity and medical cost of our patient's delay in care is a strong argument to provide more accessible healthcare to remote regions.

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Acute Portal Vein Thrombosis Diagnosed with Point-of-care Ultrasonography

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Abdominal pain is the most common presenting complaint to the emergency department (ED);1 however, acute portal vein thrombosis is an uncommon cause of abdominal pain. In the following case report, we present a patient who presented to the ED with symptoms of gastroenteritis but was ultimately diagnosed with acute portal vein thrombosis by point-of-care ultrasound (POCUS). [Clin Pract Cases Emerg Med. 2017;1(1):50–52.]

INTRODUCTION

Abdominal pain is the most common presenting complaint to the emergency department (ED) according to the National Hospital Ambulatory Medical Care Survey, with 11.1 million visits per year in 2011 with the highest rate among females age 25-44.[1] There are many different etiologies for abdominal pain and the job of the emergency physician is to differentiate serious and life-threatening causes from more benign etiologies. Portal vein thrombosis (PVT) is an uncommon cause of abdominal pain in the ED.[2] Most PVTs found are chronic and are an incidental finding on computed tomography (CT) or Doppler ultrasonography. However, PVTs can also occur acutely and usually present with more symptoms than chronic PVTs. In the following case report we present a patient who was diagnosed with acute PVT by point-of-care ultrasound (POCUS), and on further imaging was found to have extension into the superior mesenteric vein with subsequent ischemic small bowel.

CASE REPORT

A 36-year-old female presented to the ED complaining of five days of nausea and vomiting. She developed diarrhea one day prior to her visit and had acutely worsening diffuse abdominal discomfort. She had been seen in clinic four days prior and was diagnosed with suspected viral gastroenteritis. She was given ondansetron and encouraged to drink fluids to rehydrate. The patient denied smoking, and consumed alcohol occasionally. Her only outpatient medications were cetirizine for seasonal allergies and levonorgestrel-ethinyl for contraception. Upon presentation her blood pressure was 134/100mmHg, she was tachycardic (110 beats/minute), tachypneic (29 breaths/minute) and afebrile (97.5rile On exam she was noted to have mild diffuse abdominal tenderness without rebound or guarding, and there was no hepatosplenomegaly. The patient's abdomen was not distended, there was no fluid wave concerning for ascites or distended abdominal wall vessels. Initial laboratory data showed an aspartate aminotransferase (AST) of 116U/L and alanine aminotransferase (ALT) of 267U/L. Her alkaline phosphatase was unremarkable at 96 U/L. Total bilirubin was 1.8mg/dL. Her lipase was unremarkable. She had marked leukocytosis with WBC of 21.6 bil/L and left shift with neutrophils 15.4 bil/L, and a lactate of 3.1 mmol/L. Given the elevation in liver function tests, a POCUS was performed to investigate for gallbladder pathology using a Zonare C6-2 curved array probe. The POCUS showed free fluid in the abdomen and bowel wall thickening, as well as an echogenic focus in the portal vein without color flow consistent with PVT (Figures 1-3). The patient was taken directly for computed tomography (CT) instead of a more comprehensive radiology-performed ultrasound, as she appeared to have complications secondary to the PVT given the abnormal appearance of her bowel on POCUS. The CT showed a small amount of perihepatic ascites, a large filling defect within

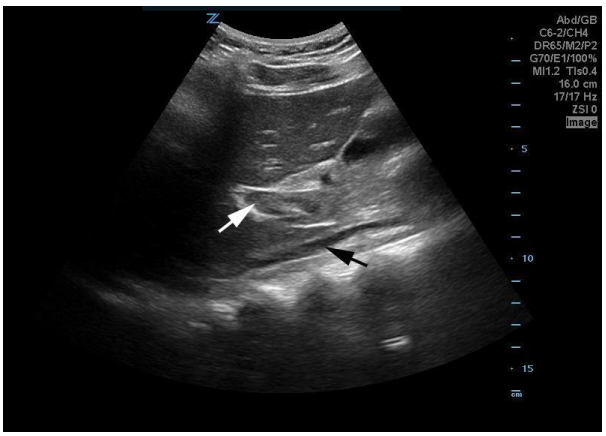

Image 1. Subcostal oblique parasagittal gray-scale image demonstrates an echogenic focus in the portal vein (white arrow), consistent with clot and a small inferior vena cava (black arrow). *IVC,* inferior vena cava

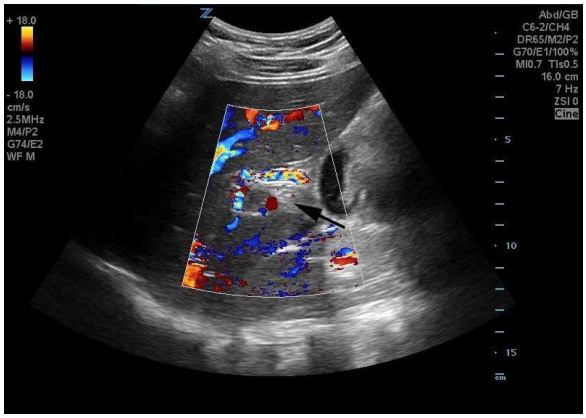

Image 2. Subcostal oblique parasagittal image with color flow Doppler demonstrates a lack of color flow in the portal vein (black arrow).

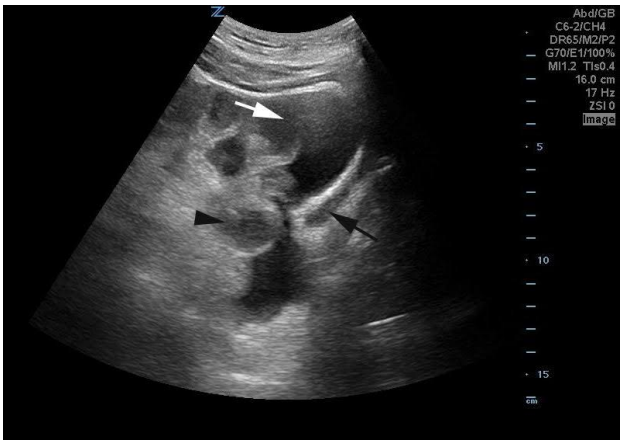

Image 3. Sagital suprapubic image demonstrates free fluid within the pelvis (white arrow) with a small bladder (black arrow) and thickened bowel (black arrow head), concerning for ischemic bowel.

the portal vein and diffuse small-bowel wall edema and mild dilation of the duodenum suspicious for ischemia.

General surgery was consulted, heparin was initiated, and fluid resuscitation was continued with a resultant decrease in her lactate to 2.2 mmol/L. The patient was admitted to the surgical intensive care unit with plan for surgery the following day. A radiology-performed ultrasound confirmed the PVT, with extension into the proximal splenic vein. Despite anticoagulation and aggressive intravenous fluid resuscitation, the patient's condition deteriorated overnight and she was noted to have an increasing lactate to 3.7 mmol/L. She was subsequently taken emergently to the operating room for an exploratory laparotomy. Interventional radiology was present and performed a mechanical and pharmacologic thrombectomy. A 35 cm area of ischemic small bowel was then resected and anastomosis was performed two days later. The patient was discharged 12 days after initial presentation on warfarin and off oral contraceptives. Initial hypercoagulability work-up showed elevated anticardiolipin IgM titers suspicious for anticardiolipin antibody syndrome; however, the confirmatory tests were negative.

DISCUSSION

PVT is an uncommon cause of abdominal pain, with an overall incidence of 1%.[2] It is most often a chronic condition that is found incidentally in the ED with Doppler ultrasonography or CT. However, PVT can also occur acutely. Acute presentations are normally symptomatic with patients complaining of abdominal pain, diarrhea and ileus. Common complications of PVT include ischemic hepatitis, variceal bleeding and, infrequently, small bowel ischemia.[3]

Due to the rarity of PVT there are few large studies exploring its complications and risk factors. The incidence, however, is known to increase with a number of conditions, most significantly cirrhosis and hepatobiliary malignancy. Other factors that increase the risk of PVT include abdominal infections, inflammatory disease, myeloproliferative disorders, coagulopathies and use of oral contraceptives.[2,4]

The presentation, diagnosis and associated complications of PVT differ based on whether the clot is acute or chronic, isolated to the portal vein, or if there is extension into the mesenteric or splenic veins. Isolated PVT,is often asymptomatic and has been found to rarely cause acute ischemia. In one study only 39% of patients with isolated PVT reported symptoms, most commonly abdominal pain. Patients with extension of the clot into the mesenteric vein reported symptoms in 92% of cases and 45% were found to have intestinal infarction.[4] Patients with acute PVT are also more likely to complain of nonbloody diarrhea. They often have small-volume ascites, systemic inflammatory response syndrome and peritonitis. Although patients with chronic

PVT can develop ischemia, they are more likely to present with hematemesis or melena due to the development of portal hypertension and variceal hemorrhage.[3]

The percentage of patients with PVT with extension into the mesenteric vein has been estimated to be as high as 55%.[5] Presence of thrombosis in the mesenteric vein was also associated with higher mortality rates ,estimated from 20 to 50%.[6] The duration of the thrombosis in the mesenteric vein also affects mortality rates. Patients with acute clots have a three-year survival rate of 36% versus 83% for those with chronic thromboses.[7] Isolated mesenteric venous thrombosis, without concurrent portal vein clot, have also been reported. Patients presenting with isolated mesenteric thrombosis are a particular challenge to clinicians as both ultrasound and CT, with combined sensitivity of only 50%, often fail to visualize the clot as compared to a 97% combined sensitivity when the thrombosis extends into the portal or splenic vein. If there is high suspicion, angiography remains the gold standard for diagnosis.[8]

Ultrasound has been used to diagnose PVT for decades, but given that up to 30% of patients with PVT will not have an echogenic thrombosis on gray-scale US it was not until the advent of color Doppler imaging that it became a reliable test.[9] With color Doppler, clinicians are able to visualize flow noninvasively. When compared to angiography or surgery, color Doppler US has a sensitivity of 89% and specificity of 92%.[9] Characteristics of PVT on color Doppler include increased arterial flow in patient with low portal flow, postprandial reversal of portal flow from hepatopetal to hepatofugal, and development of collateral circulation.[10] Acute and chronic thrombosis appear differently with color Doppler. In both cases an echogenic thrombus is often but not universally seen. Chronic PVT more often will show cavernous transformation of the portal vein with collateral formation.[9,10]

Both acute and chronic PVTs are typically treated with oral anticoagulants. However, if there is extension of the clot into the mesenteric vein with evidence of ischemia, more invasive measures, including surgery and thrombectomy, may be indicated. This case shows the importance of keeping PVT on the differential for patients presenting with abdominal pain. It also shows that POCUS with color Doppler may speed the diagnosis of this potential life-threatening condition.

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Progressive Disordered Movements in an Infant Leads to Rare Diagnosis

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Desmoplastic infantile ganglioglioma (DIG) is a supratentorial superficially-located cystic neuroepithelial tumor. It is an exceedingly rare tumor with an incidence of <0.1% of central nervous tumors; approximately 60 cases have been reported in the literature. We present a case of a three-month-old infant with progressive disordered movements described as intermittent upper body stiffening with associated eye blinking, drooling, and change in level of alertness. A seizure was witnessed in the emergency department, after which the child was sent for imaging studies. Magnetic resonance imaging (MRI) revealed a large solid and cystic mass in the temporal region measuring 8.6cm x 7.9cm x 5.1cm. The infant underwent complete surgical resection, and post-surgical pathology revealed a diagnosis of DIG. The patient had an excellent post-operative course in the months following discharge. At his last well-child visit, no neurological deficits were appreciated and the infant was meeting expected milestones for his age. [Clin Pract Cases Emerg Med. 2017;1(1):53–55.]

INTRODUCTION

Desmoplastic infantile ganglioglioma (DIG) is a supratentorial superficially located cystic neuroepithelial tumor. It is an exceedingly rare tumor with an incidence of <0.1% of central nervous tumors, and there have been approximately 60 cases described in the literature.[1] It commonly presents with increasing head circumference, signs of hydrocephalus, setting sun sign (upward gaze paresis), or, less commonly, seizure activity.[2] Less common presentations include repeated vomiting, developmental delay, and oculomotor defects; however, sensory and motor deficits are rare.[2] Here we describe a case of a three-month-old male infant who presented to the emergency department (ED) with a history of "jerking" movements and stiffening of the upper body, worsening over the course of a few weeks. ED work-up revealed a large intracranial mass. Subsequent inpatient evaluation and surgery showed the mass to be a DIG.

CASE REPORT

A three-month-old full term male was brought to the pediatric ED by his grandmother for evaluation of "jerking" activity worsening in frequency and severity for approximately three weeks. The grandmother described the "jerking" activity as intermittent upper body stiffening with associated eye blinking and drooling with a change in level of alertness just following the event. She reported that the episodes had increased in frequency over the few days prior to ED arrival but claimed the episodes had been present for the approximately one month. The grandmother stated that initially the jerking occurred approximately once per day; however, there were three episodes noted on the day of presentation. She described these episodes as lasting seconds to minutes and were not associated with color change, respiratory distress, or spitting up. They occurred at any time of the day, with no predilection for early morning or sleep arousal, and there was no association with feeding. She denied any recent history of falls, head trauma, fever, cough, congestion, diarrhea or rash. The remainder of the review of systems was negative. The infant was formula-fed every four hours and had been producing a normal amount of wet diapers and stool. When questioned about the pregnancy, the grandmother was

unsure if the mother had been using drugs or alcohol, but stated that the infant was the product of an uncomplicated labor and delivery with a normal newborn screening. His birth weight was reported as 2.86 kg. The infant attended daycare, but there were no known sick contacts. The grandmother stated that the baby had been healthy, meeting his development milestones, and gaining weight appropriately according to his last well-child visit. There was a maternal history of multiple sclerosis reported by the grandmother.

On physical exam, the infant appeared well and was noted to make eye contact with the grandmother. Initial weight and vital signs reported at triage were as follows: weight: 5.51 kg, temperature 98.0º F, respiratory rate 22 breaths/min, heart rate 143 beats/min, oxygen saturation of 97% on room air. The pertinent positives of his exam included a full anterior fontanelle but no bulging. Extraocular movements were equal with no nystagmus appreciated. There was a mild left ocular prominence noted but no proptosis. He had no facial deformity or external signs of trauma with tears present and moist mucous membranes. His neck was supple, and appropriate head control was appreciated when he was seated in his grandmother's lap. An overall slight increase in his tone was noted. All movements were observed to be symmetrical with no obvious motor deficit or weakness.

Moments after the initial assessment, the ED staff was called to the bedside by the grandmother. The infant was observed to have left eye blinking followed by asynchronous right eye blinking with drooling at the mouth. He was unresponsive to direct confrontation. No other jerking motions were observed. The episode lasted less than two minutes during which time he was placed on a cardiac monitor and vital signs were stable. Immediately following the episode, the infant was not responding normally to the grandmother but became increasingly more alert and returned back to baseline within several minutes.

Given his initial presentation and witnessed seizure activity, laboratory tests and imaging were ordered. The results of his lab work were as follows: WBC 10.4 x 10^3, Hgb 12.4 g/dL, Hct 36.3%, platelets 302 x 10^3/mm;^{3} Differential: lymphocytes 77%, neutrophils 14%, eosinophils 9%. Sodium 136 mEq/L, potassium 5.5 mEq/L, chloride 106 mEq/L, bicarbonate 20 mEq/L, BUN 5 mg/dL, creatinine 0.22 mg/dL, glucose 100 mg/dL, magnesium 2.1 mEq/L, phosphorus 5.5 mg/dL, anion gap 10.

In an attempt to spare exposure to ionizing radiation, the decision was made to perform a stat non-contrast magnetic resonance imaging (MRI) of the brain. After review of the scout images from MRI, a magnetic resonance angiogram protocol was added. Imaging revealed a large supratentorial cystic and solid mass in the left temporal region measuring 8.6cm x 7.9cm x 5.1cm. A plan was then made to transfer the patient to a pediatric intensive care unit, which could afford a higher level of care. A loading dose of levetiracetam (55 mg) intravenously was administered and D10 NS was run at maintenance.

The infant had five seizures on the day of admission, two of them witnessed. He was given an additional dose of levetiracetam 20 mg/kg IV and then continued on a maintenance dose of 10 mg/kg. The patient was also started on a dexamethasone 0.5 mg/kg. No subsequent seizure activity was reported following medication titration. Operative management was conducted by pediatric neurosurgery on hospital day 6. The patient underwent a left craniotomy with gross tumor resection. Pathology report indicated the tumor was consistent with DIG. In the post-operative period, the patient was observed to have a left lateral gaze palsy, and repeat imaging revealed a new acute infarct of the right thalamus. Due to the acute infarct, a neurology consult was obtained and the infant underwent a full work-up for a new-onset hypercoaguable state. The infant was found to have a slightly elevated anti-thrombin III level, and the infarct was not attributed to surgical management. Echocardiography was found to be normal and the patient was subsequently started on aspirin. The infant's subgaleal Jackson-Pratt drain remained in place until post-operative day 3. No respiratory or gastrointestinal complications arose and the patient was tolerating full feeds within several days post-operatively.

Despite the right thalamic infarct, the patient had an excellent post-operative course in the months following discharge. At his last well-child visit, no neurological deficits were appreciated and the infant was meeting expected milestones for his age.

DISCUSSION

DIG is a supratentorial superficially-located cystic neuroepithelial tumor. It is characterized by prominent desmoplasia with neoplastic glial component or neoplastic glioneuronal component.[1, 2] It is an exceedingly rare tumor with an incidence of <0.1% of central nervous tumors. Approximately 60 cases have been reported in the literature. It primarily occurs within the first 24 months with a slight predilection for males over females (1.7:1).[1] It is a benign tumor recognized by the World Health Organization (WHO) under the category of glio-neuronal tumors and is classified by the WHO as a grade I tumor.[3, 4]

Clinical presentation is usually consistent with increasing head circumference, signs of hydrocephalus, setting sun sign or, less commonly, seizure activity. Approximately one in four children are diagnosed following a seizure.[2] Less common presentations include repeated vomiting, developmental delay, and oculomotor defects; however, sensory and motor deficits are rare.[2]

The supratentorial region is preferentially involved, especially the frontal and parietal lobes, followed by the temporal lobe.[1, 3] The large size of these tumors and the relative lack of obtundation in these children suggest that they are very slow growing and likely congenital in origin.[5] MRI is the most useful diagnostic tool. Typical imaging reveals a large supratentorial tumor often involving more

than one lobe, with both large cystic and solid components with firm attachment to the dura.[2] A recent study suggested that diffusion-weighted imaging may be especially helpful in differentiating DIG from other tumors such as infantile glioblastoma multiforme.[6] Contrast enhancement was reported to be homogenous and avid in DIG tumors, whereas infantile glioblastoma tumors had more heterogeneous uptake.[6] Radical surgical resection is the treatment of choice in DIG. Overall, there is a very good prognosis with complete resection.[1,2,5] However, because the mass may be firmly affixed to the dura or may penetrate into deeper cortical layers of the brain this may sometimes be difficult to accomplish, in which case adjunctive chemotherapy or radiation therapy may be used.[2,6]

CONCLUSION

The differential diagnosis in a three-month-old infant presenting to the ED with new onset seizures is broad. Several important considerations include child abuse, trauma, mass lesions, inborn errors of metabolism, electrolyte disturbance including hypoglycemia and sodium derangements, subtle seizures, jitteriness, infantile spasms (West syndrome), benign myoclonic jerks, and migrating partial epilepsy in infancy. Febrile seizures are only considered in infants greater than six months of age. If fever does occur with seizure in a child younger than this, it is a harbinger of a more serious underlying etiology. Our case was a three-month-old infant with worsening intermittent generalized seizures of unknown origin. MRI revealed a large, superficially located solid and cystic mass in the left parietal lobe. Pathology of the tumor following total resection revealed the diagnosis of desmoplastic infantile ganglioglioma.

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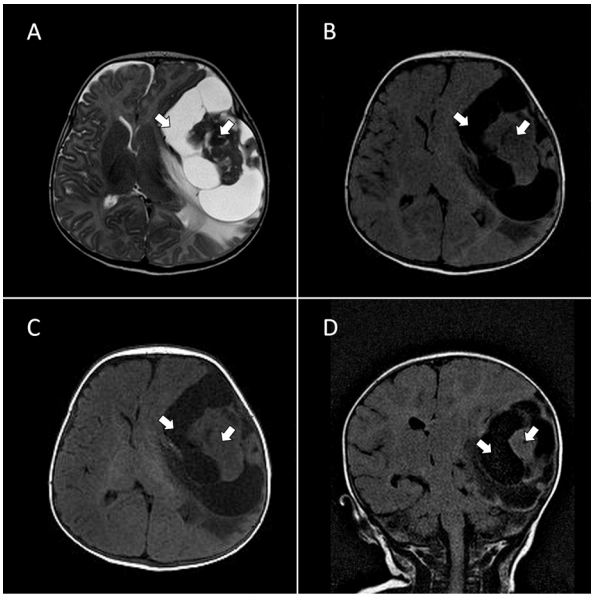

Image. MRI revealing an 8.6cm x 7.9cm x 5.1cm semi-cystic-semi-solid mass (arrows) with epicenter in the left temporal region. T2-weighted axial view (A). T2-weighted with flair axial view (B). T1-weighted axial view (C). T2-weighed with flair coronal view (D). *MRI,* magnetic resonance imaging

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Fecal Impaction With Multisystem Organ Involvement

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Fecal impactions are a common complaint in the emergency department (ED) population. The potential for significant derangement in physiologic processes of other organ systems is often underappreciated. A 19-year-old male, previously healthy, presented to the ED at our institution with complaint of abdominal pain, which was found to be secondary to severe fecal impaction. In the search for alternative diagnoses, imaging was performed, which revealed effects on multiple other organ systems. This case illustrates the secondary effects of a severe fecal impaction. The emergency physician must be aware of these consequences, as the opportunity to review labs and imaging is not often provided in the standard work-up of these patients. [Clin Pract Cases Emerg Med. 2017;1(1):56–58.]

INTRODUCTION

Fecal impaction is a common complaint addressed in the emergency department (ED). Serious complications are not often observed, and the problem is commonly resolved quickly with a manual disimpaction. Most patients do not require laboratory evaluation or advanced imaging.[1] Occasionally the search for alternative diagnoses requires diagnostic testing beyond the history and physical exam. Imaging may demonstrate secondary diagnoses related to the physiologic disruption caused by severe retention of fecal material.

CASE REPORT

A 19-year-old male presented to our institution complaining of abdominal pain for two weeks. He reported that he initially felt constipated and started taking over-the-counter medications to self-treat his condition. These medications included polyethylene glycol, various stool softeners, and a colonoscopy preparation product he received from a family member. One week prior to presentation, he developed watery stools and discontinued all medications. Since that time he reported continued watery, non-bloody stools of small volume approximately twice daily. The patient noted a remote history of constipation as a child, requiring manual disimpaction. Since that time, he had been taking polyethylene glycol daily without further issues. There was no personal or family history of Hirschsprung's disease. He denied any nausea, vomiting, or fevers. On further history taking he did admit to using his family member's oxycodone over the prior several weeks. There was no recent antibiotic use and travel history was unremarkable. Physical examination was significant for a distended and firm abdomen, hypoactive bowel sounds, and mild generalized tenderness to palpation without peritoneal signs. Rectal exam revealed a small amount of brown watery stool externally and hard intraluminal stool in the proximal rectum felt with the fingertip. Fecal occult blood testing was negative. The patient complained of severe rectal pain, limiting further evaluation of the rectal vault more proximally.

Laboratory values were notable for creatinine 1.03 mg/dL, total bilirubin 1.1 mg/dL, and direct bilirubin 0.29 mg/dL. The remainder of the complete metabolic panel was within normal limits, as was a complete blood count and urinalysis. Due to the hard, distended abdomen on exam, there was concern for the possibility of serious intra-abdominal pathology.

It was felt that computed tomography of the abdomen and pelvis would provide the necessary information to rule out alternative diagnoses, as well as further delineate the location of a possible bowel obstruction. Imaging revealed extensive retained fecal material, which distended the sigmoid colon up to 18.2 centimeters and extended into the upper abdomen, exerting mass effect on multiple solid organs (Image 1). Bilateral hydronephrosis was observed secondary to mass compression of

bilateral ureters (Image 2). The bladder was displaced anteriorly and superiorly from its expected position. In addition, the colon can be seen compressing the left hepatic lobe (Image 3).

Colorectal surgery was consulted and initially attempted manual disimpaction in the ED. However, this was aborted secondary to severe patient discomfort. Due to the extensive nature of the impaction, treatment with naloxone was not considered a viable option. The risk of bowel perforation discouraged further attempts at relieving the impaction under conscious sedation at the bedside. Thus, the patient was taken to the operating room for manual disimpaction under general anesthesia. The large stool ball in the proximal rectum needed to be broken into pieces and removed manually. Greater than 15 pounds of soft stool were evacuated. The patient was admitted for observation and was discharged the following hospital day with instructions to follow up.

DISCUSSION

Constipation is a common complaint in ED patients. The prevalence of constipation in the community is estimated to be around 16%.[1] Constipation may be attributed to stool quality, colonic motility, or outlet obstruction. As symptoms progress and become more severe, patients may develop obstipation. This can further develop into a large bowel obstruction, which can be complicated by perforation if not recognized and treated expeditiously.

Colonic contractions normally propel stool several times a day, often postprandially. It is known that voluntary suppression of defecation is a risk factor for constipation due to decreasing bowel motility and a blunted motor response to eating.[2] Additionally, a number of other etiologies and contributing factors to constipation exist, including low fiber intake, dehydration, spinal cord injury, stroke, dementia, advanced age, and Hirschsprung's disease. Constipation is a common medication side effect and is associated with opiate analgesics, anticholinergics, antacids, iron, and calcium-channel blockers. Often, fecal impaction is a consequence of chronic or untreated constipation.

Evaluation of the patient with possible fecal impaction should start with a detailed history, including special attention to the risk factors described above. A key element in the patient interview is eliciting a history of previous impaction, which is found in up to 39% of patients.[3] Physical exam findings may include signs of dehydration, diffuse abdominal tenderness, and fullness in the left lower quadrant corresponding to fecal matter collecting in the rectosigmoid colon.

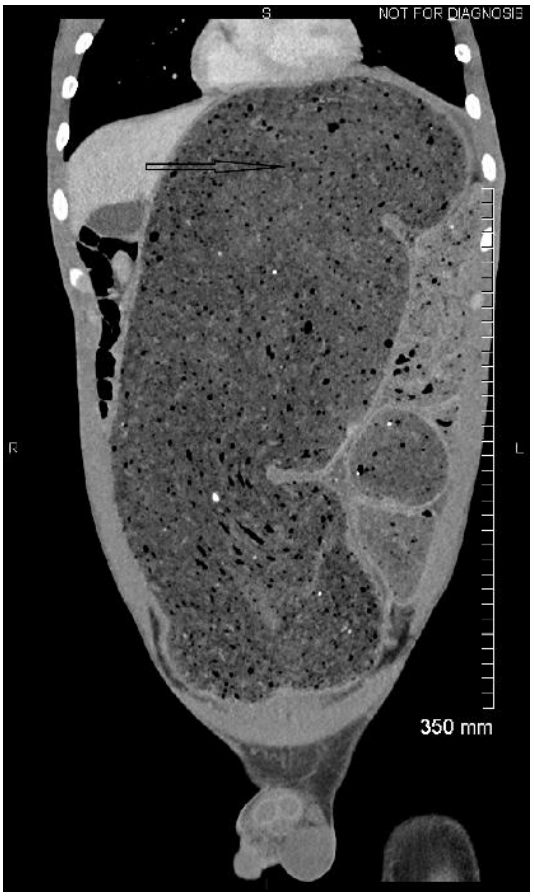

Image 1. Coronal section of the abdomen by computed tomography displaying extent of the extensive stool burden.

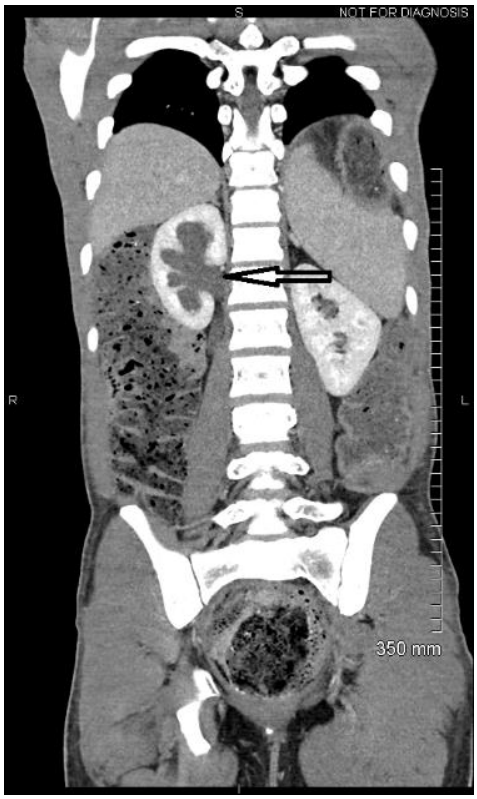

Image 2. Coronal section showing right hydronephrosis (arrow) due to obstructive uropathy from fecal material.

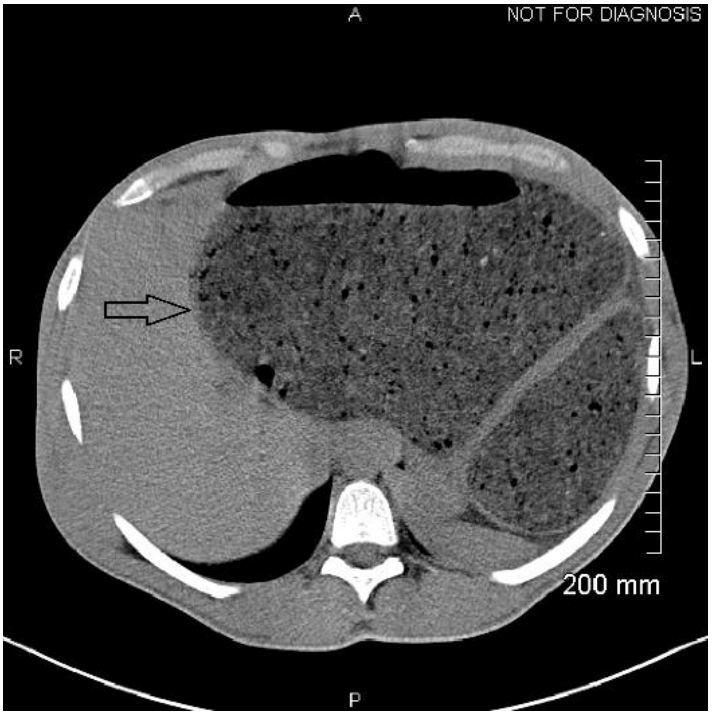

Image 3. Distended colon exerting mass effect on the liver (arrow).

A wide array of complications attributed to fecal impaction has been noted in the literature, primarily published as case reports. A recent systematic review detailed the relative proportions of these complications.[4] The most common complications observed were secondary to local compression of the colon and included intestinal perforation, obstruction, and ulceration. Interestingly, a significant number of cases involved complications without direct gastrointestinal damage. The highest number of cases involved obstructive uropathy, which was observed in our patient. Additional cases noted urinary bladder damage, as well as compression of nerve and vascular structures. Although our patient was found to have hepatic compression with mild hyperbilirubinemia and ascites, the effect on other organ systems has been documented in the literature. Consideration of these uncommon but serious medical conditions should be a part of clinical decision-making in these patients. The recognition of the secondary effects is important, in that these effects must be followed to resolution to exclude underlying pathology in those organ systems that may be obscured by the fecal impaction.

Emergency physicians are trained to consider the most serious diagnoses in their differential decision- making first. Constipation with fecal impaction is often thought to be a benign condition; however, our case demonstrates that fecal impaction can have effects on multiple organ systems. Despite our patient's young age, there was observable impact on both his renal and hepatic systems. The patient recovered quickly without any known lasting morbidity; however, the consequences may be more significant in patients of advanced age or with other comorbid conditions. It is important for the emergency physician to be aware of these findings and consider additional work-up in patients with high risk of complications. Additionally, ensuring appropriate follow-up with a primary care physician and instituting an appropriate bowel regimen can prevent further instances and subsequent complications.

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Pediatric Oral Commissure Burn

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INTRODUCTION

The labial, or oral, commissure is the site of a distinct pediatric injury that commonly presents as the result of an arc of electricity, which can preferentially injure the mucous membranes due to the surrounding electrolyte-rich saliva and the relatively low resistance of the tissues. The injury frequently involves a power cord of some type either being bitten or sucked on by the patient. The management of this injury can have profound impact on the aesthetic and functional outcomes for the patient.

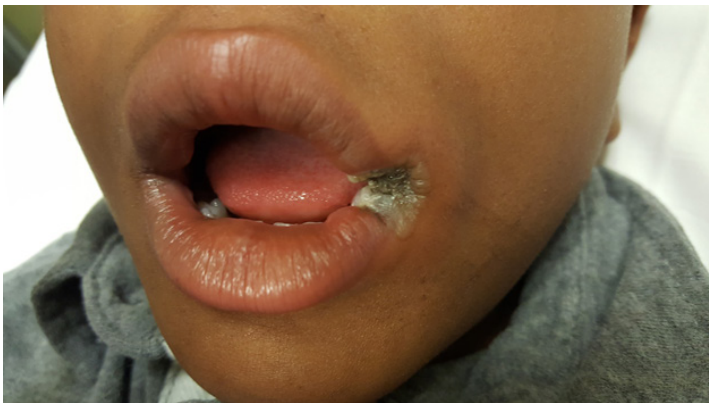

Image. Left oral commissure burn with tissue destruction, eschar formation, and surrounding erythema in pediatric patient.

CASE REPORT

A five-year-old male presented to the emergency department at 2 a.m. after having bitten through a television power cable and suffering a burn injury to his mouth 20 minutes prior to arrival. The patient denied any other injuries and located pain only to the left side of his lips. He denied tongue pain.

Physical exam showed a gray and white eschar to the left oral commissure without any evidence of current or recent bleeding. Intraoral mucosa was only involved near the commissure and the tongue was uninjured. There was mild erythema and induration of the facial skin surrounding the eschar. The remainder of the exam was normal.

Acetaminophen was given for analgesia; however, no active intervention was required for the burn or resultant eschar. The patient was admitted to the general pediatric unit overnight for monitoring and pain management before being discharged the following day. He was referred to a regional burn specialist for non-emergent consultation within one week.

DISCUSSION

Emergent management of an oral commissure burn depends on the extent of the injury. Evidence of airway involvement with singed nose hairs, soot-colored mucous, wheeze, stridor, voice changes or coughing may require definitive airway management with endotracheal intubation. General burn treatment principles such as the "rule of nines," palmar surface area method, and Parkland formula, will guide treatment in those more extensively burned.

Oral commissure burns are classified by a system proposed by Al-Qattan et al which uses depth and location to guide treatment and prognosticate aesthetic and functional outcomes. In our case, the patient would be considered in the moderate category where splinting would be the recommended treatment of choice with the possibility of the need for commissuroplasty.[1] Timing and technique of definitive operative management of oral commissure burns remains a controversial topic,[2] making consultation with an expert in pediatric burn management crucial from the emergency physician's perspective. Bleeding at the burn site is initially uncommon, though it may be seen between one and two weeks after the injury in up to 25% of cases. It is associated with exposure of the damaged labial artery with the slough of the eschar.[2]

Parents should be instructed on this point as well as to apply direct pressure to any bleeding.

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Massive Emphysematous Pyelonephritis

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INTRODUCTION

A 58-year-old male presented to an outside hospital with altered mental status and right flank pain for three days. Septic work up, including computed tomography of the abdomen and pelvis, were significant for diabetic ketoacidosis, pyelonephritis, and significant air replacing much of the right kidney, consistent with emphysematous pyelonephritis (Image). The patient was transferred to our facility for a higher level of care.

The patient was stabilized, given intravenous (IV) antibiotics, and admitted to the intensive care unit with a diagnosis of septic shock secondary to emphysematous pyelonephritis.

DISCUSSION

Our case presents an image of a condition that is rare and particularly severe, as shown by free air not only in the right renal parenchyma, but also extending outside the capsule, around the renal vasculature, and into the left peri-renal space.

Emphysematous pyelonephritis is a relatively rare infection, seen only 1-2 times per year in a typical busy urological department in the United States. It affects patients with diabetes in 95% of cases. *E. coli* and *Klebsiella* account for over 90% of cases, although *Proteus mirabilis*, *Pseudomonas*, and *Streptococcus* are also seen. Gas accumulates due to rapid necrosis of the renal parenchyma and peri-renal tissue, as

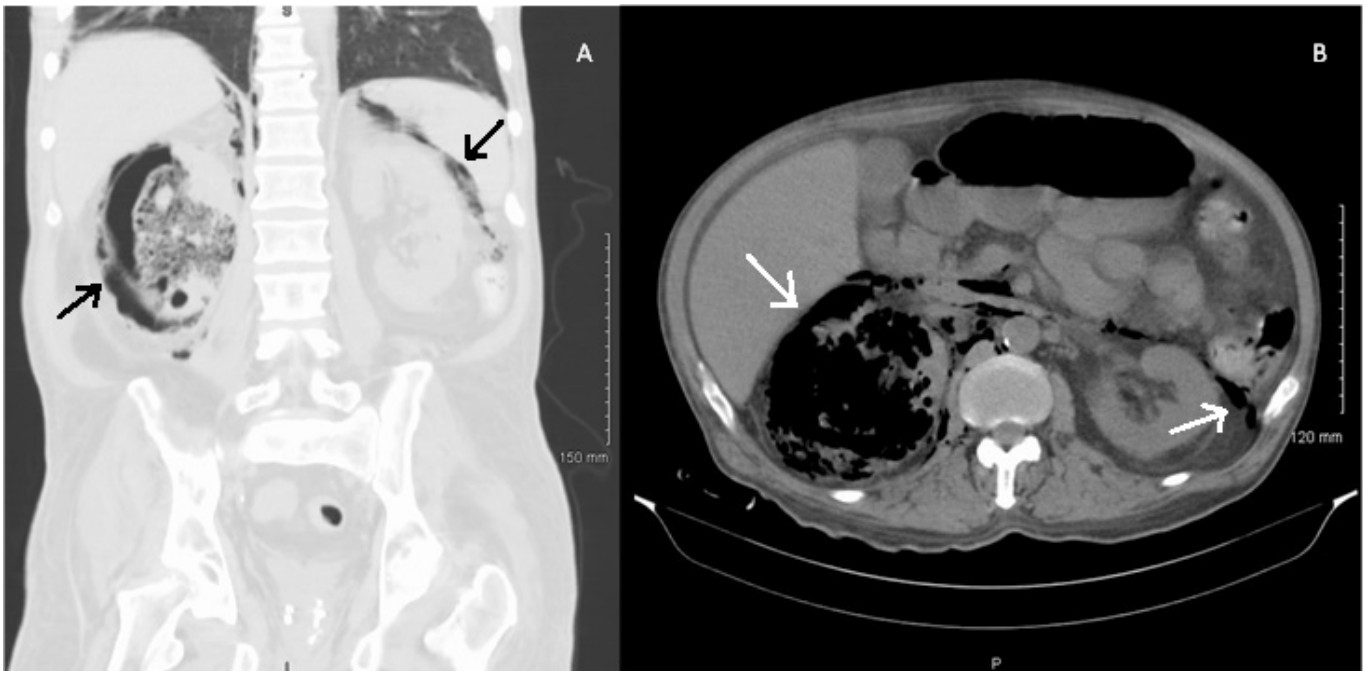

Image. A. Coronal view of a computed tomography (CT) of the abdomen and pelvis, in the lung window, showing bilateral emphysema. B. Axial view of CT of the abdomen and pelvis, without contrast, showing emphysema replacing the right kidney.

opposed to gas appearing as a byproduct of anaerobic bacteria as is the case in necrotizing fasciitis. The condition is fatal if not treated appropriately, and the mainstay of treatment is nephrectomy in conjunction with IV antibiotics for severe, disseminated infection.

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Flagellate Dermatitis: A Culinary Flogging

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CASE

A 30-year-old male, previously healthy, presented with a rash of three days duration. Lesions were first noticed on his right thigh, chest and back. The rash was intensely pruritic, but not painful. The patient tried self-treatment with oral diphenhydramine but had minimal relief. Review of systems was negative. He denied any sick contacts, recent travel, outdoor, chemical or other irritant exposures. Past medical history was noncontributory; the patient denied previous rashes. Exam was notable for a generalized rash in centripetal distribution. Lesions were raised, erythematous, and ranging in appearance from papules to wheals. Most striking was the crisscrossed linear pattern (Image 1a, 1b, 1c). The

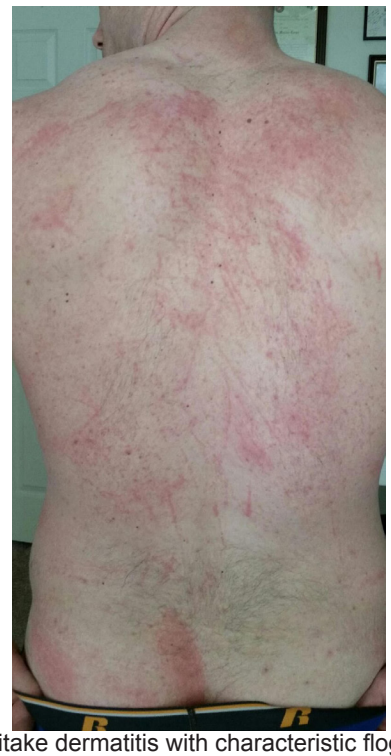

Image 1a. Shiitake dermatitis with characteristic flogged appearance.

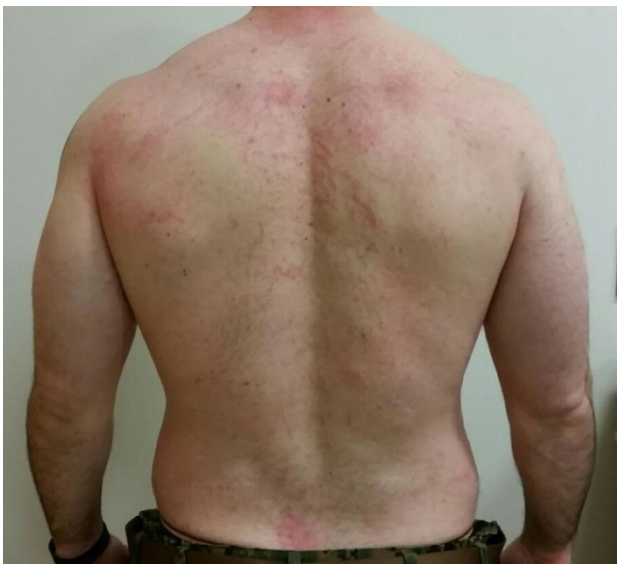

Image 1b. Sequential photo on day 2 after initiating steroid burst.

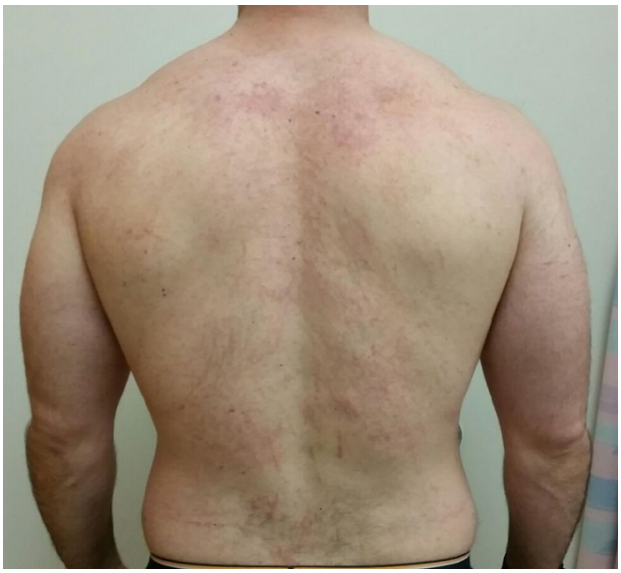

Image 1c. Sequential photo on day 5 after initiating steroid burst.

patient was initially treated for allergic reaction with topical hydrocortisone and oral hydroxyzine. He presented again two days later with worsening rash affecting knees, groin, and elbows, in a similar appearance. A detailed exposure history revealed consumption of multiple dishes containing Shiitake mushrooms 48 to 72 hours prior to onset at a restaurant serving Chinese cuisine. Furthermore, the patient had continued to eat leftovers after initial presentation.

Flagellate dermatitis, alternatively shiitake dermatitis, was diagnosed with positive history of ingestion of shiitake mushroom, *Lentinus edodes*. Shiitake is becoming more popular as a culinary ingredient not limited to Asian cuisine. Flagellate dermatitis is named for the flogged-like appearance, consistent with previous case reports.[1] Close examination of the rash revealed a confluence of small papules into the linear streaks. Reports have been associated with ingestion of undercooked or raw mushroom.[1] Pathophysiology remains poorly understood with evidence for both toxic reaction to lentinan polysaccharide and allergic-type hypersensitivity reaction.[2] Biopsy and skin prick testing have been nonspecific and not validated for routine testing.[3] Improvement was seen after similar treatment used in a previous case consisting of oral prednisone 50mg burst, oral cetirizine 10mg daily and topical mometasone furoate 0.1% daily.[4] The rash resolved in approximately 10 days from start of treatment.

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Traumatic Right Ventricular Rupture after Blunt Cardiac Injury: CT Diagnosis after False Negative Pericardial Window on FAST Due to Concomitant Pericardial Rupture

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CASE

A 93-year-old male presented to a Level I trauma center in hemorrhagic shock after a head-on motor vehicle collision in which he was a restrained driver. Initial vitals were temperature 35ºC, BP 100/50 mmHg, HR 63 beats/minute, respirations 28/minute, and oxygen saturation of 93% on 10L. Physical exam was significant for Glasgow Coma Scale 14 (-1 for confusion), decreased breath sounds bilaterally with diffuse chest wall tenderness, a seatbelt sign in the driver-side chest and hip distribution. Cardiac exam was without murmur. Bedside focused assessment with sonography for trauma (FAST) was negative, and chest radiograph revealed multiple bilateral rib fractures and large right hemothorax. The patient developed worsening hypotension after a chest tube yielded >1L of gross blood despite blood product resuscitation. Computed tomography of the chest revealed multiple thoracic injuries including but not limited to several rib fractures, sternum fracture, large right-sided hemothorax and right ventricular rupture with leak of contrast and associated hemopericardium (Image). The patient survived an emergent thoracotomy in the emergency department for repair of the right ventricular rupture.

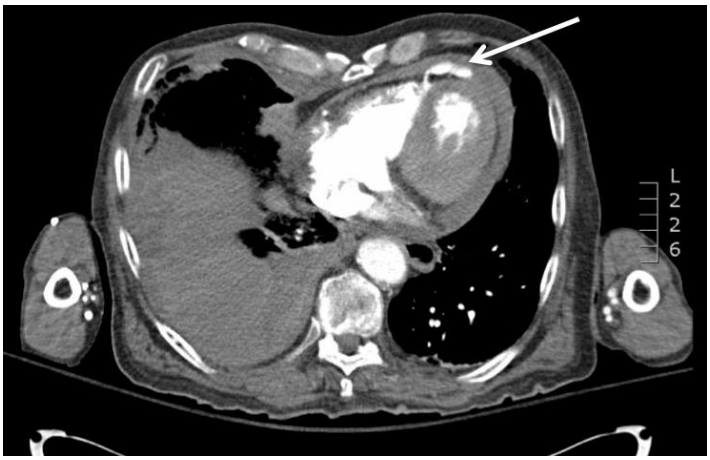

Image. Computed tomography of chest revealing rupture of right ventricle with leak of contrast and associated hemopericardium (white arrow).

DISCUSSION

Blunt cardiac injury encompasses multiple injuries, including contusion, acute valvular disorders, and chamber rupture.[1] Blunt traumatic cardiac rupture is a very rare occurrence accounting for 0.5% of blunt trauma cases with a high mortality rate.[2] We believe the initial negative FAST scan was due to a concomitant cardiac and pericardial rupture, which allowed blood to drain into the right hemithorax. Coexisting pericardial rupture in patients with cardiac rupture obscures the diagnosis and contributes to mortality.[3,4] False negative pericardial ultrasound secondary to a concomitant pericardial laceration and subsequent decompression of the cardiac hemorrhage into the ipsilateral pleural space is extremely rare and has only been recently described in the literature.[5] This case/image highlights the importance of considering an underlying cardiac injury in the presence of a negative FAST pericardial window in patients with a traumatic hemothorax.

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Orbital Trapdoor Fracture: An Open-and-shut Case?

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INTRODUCTION

A trapdoor fracture is a rare condition defined as a minimally displaced fracture of the orbital floor that has spontaneously reduced to its original position incarcerating an extraocular muscle.[1] Clinicians have described the trapdoor fracture as a "white-eyed blowout" because of a paucity of physical examination abnormalities.[2] Further complicating diagnosis, the injury may be radiographically occult on orbital computed tomography (CT).[3] Trapdoor fractures are important for the emergency physician to identify because urgent surgical repair is recommended to reduce morbidity.

CASE REPORT

A 26-year-old man presented with binocular vertical diplopia after an assault. He denied loss of consciousness, headache, or vomiting. Physical examination revealed a Glasgow Coma Scale of 15, normal visual acuity and pupils, mild periorbital ecchymosis, and restricted right ocular motion in temporal downward gaze. There was no injection of the sclera, bony step-off, enophthalmos, or proptosis. Head and orbital CT were reported as negative for injury. A maxillary mucosal retention cyst was incidentally noted on CT (Images 1 and 2).

Ophthalmology was consulted and performed forced duction testing, which confirmed gaze restriction. The patient was taken to the operating room where a trapdoor fracture with entrapment of the inferior rectus muscle was diagnosed and surgically repaired. Retrospective review of the CT revealed that the soft tissue density misdiagnosed as a maxillary sinus cyst displayed radiodensity characteristics consistent with herniation of orbital fat and entrapped muscle fibers (Images 3 and 4).

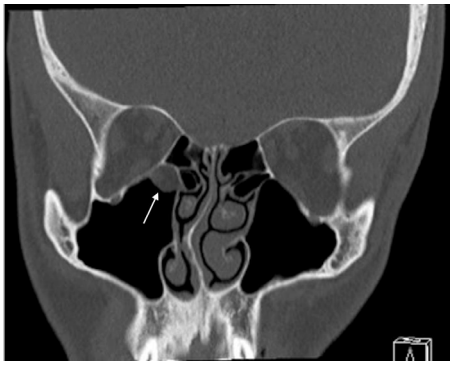

Image 1. Coronal non-enhanced computed tomography in bone window: Soft tissue density noted in the maxillary antrum and no definite fracture. Lesion initially called a mucosal retention cyst (arrow).

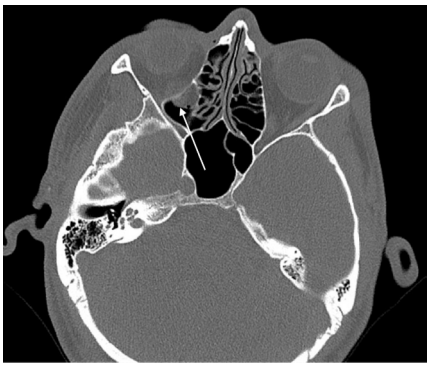

Image 2. Axial non-enhanced CT in bone window: Soft tissue density again noted in the maxillary antrum (arrow). No definite fracture.

DISCUSSION

Because of their relative bony elasticity, trapdoor fractures are reported much more frequently in children, whereas adults tend to present with readily apparent comminuted fracture patterns.[4] However, more recent literature suggests this injury is not exclusive to pediatric patients and can occur in young adults.[5-7] Trapdoor fractures may be missed unless extraocular muscles are tested through the full range of motion and close attention is paid to the location of the orbital soft tissues on CT.[3] The emergency physician must consider this diagnosis in younger patients with orbital trauma and abnormal ocular motility, even with a non-diagnostic CT, because operative intervention within

24 hours is associated with improved outcomes.[8] Reported complications of delayed surgical repair include residual gaze restriction and diplopia secondary to ischemia of the extraocular muscle.[9]

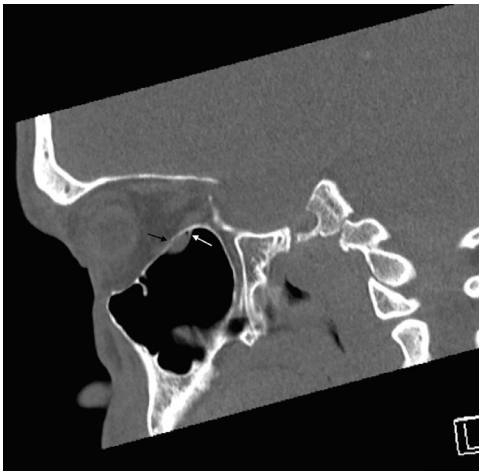

Image 3. Sagittal non-enhanced CT in bone window: Subtle defect in orbital floor (black arrow) with small focus of air adjacent to soft tissue density (white arrow).

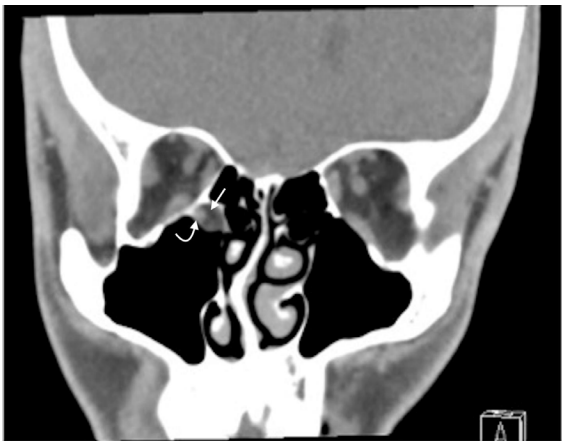

Image 4. Coronal non-enhanced CT in soft tissue window: Inferior rectus muscle (curved arrow) and orbital fat (straight arrow) in the extra-orbital maxillary antrum.

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The Daughter Cyst

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CASE REPORT

A 35-year-old woman, gravida 4 para 3 estimated at 9+1 weeks gestational age by uncertain last menstrual period, presented with vaginal bleeding. She endorsed unprotected intercourse eight weeks prior and took Plan B One-Step ("morning-after pill") within 48 hours. Quantitative human chorionic gonadotropin (hcg) resulted at 30 mIU/ml. Bedside transabdominal ultrasound was concerning for ectopic pregnancy (Video). Is this ectopic pregnancy?

Radiology performed a transvaginal ultrasound, which was negative for intrauterine or ectopic pregnancy. Obstetric consultants were uncertain if the structure seen at bedside represented an ectopic (Image). Radiology believed that this structure, which appeared initially to be an extra-uterine yolk sac, was instead a daughter cyst. The patient was well appearing and hemodynamically stable. She was discharged with ectopic precautions and a repeat 48-hour quantitative hcg, which ultimately trended to 0.

DISCUSSION

The daughter cyst sign indicates an uncomplicated ovarian cyst.[1] It is a peripherally based simple cyst within a larger simple cyst.[2] On pathology, it represents a stimulated ovarian follicle.[2] This sonographic finding must be differentiated from an ectopic pregnancy in any woman with the potential to become pregnant.[1] Previously documented cases in the literature

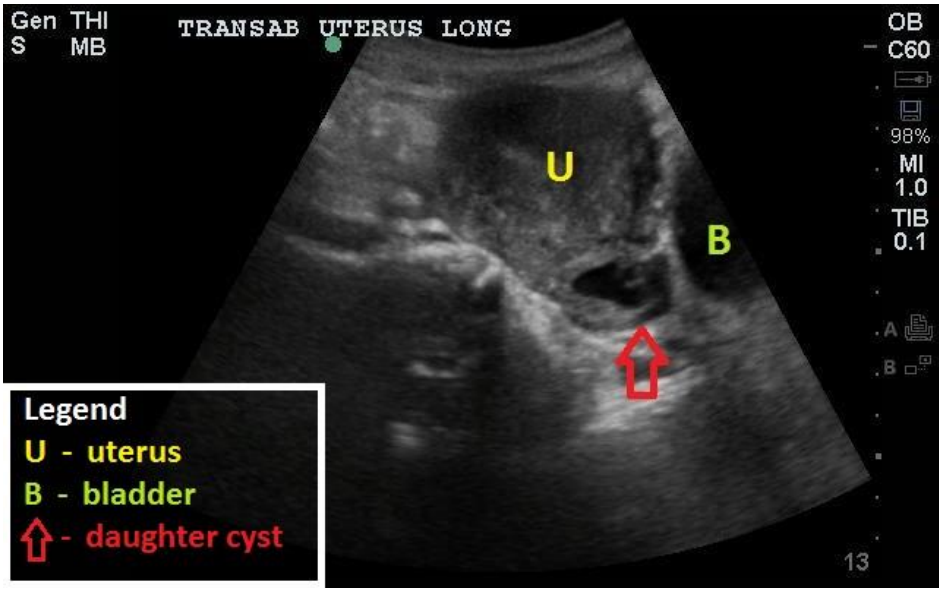

Image. Point-of-care transabdominal ultrasound demonstrating the uterus and daughter cyst in the sagittal plane.

are limited to case reports involving pediatric females with McCune –Albright Syndrome and precocious puberty, as well as fetuses with incidental cysts mimicking ectopic pregnancy.[1,2] Differentiation can be made with a quantitative hcg, which is negative in the case of the daughter cyst. Differentiation is also made sonographically with a "ring of fire" sign on the structure's periphery, indicative of increased color Doppler flow to an ectopic pregnancy, whereas a true daughter cyst has no increased flow.[1] In the case of our patient, she had a down-trending hcg due to recent administration of Plan B for unwanted pregnancy, and an incidentally noted daughter cyst that was initially concerning for an ovarian pregnancy.

Video. Point-of-care transabdominal ultrasound video clip demonstrating the uterus and daughter cyst in the axial plane.

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Point-of-care Ultrasound in Pregnancy: Think Congenital Zika Virus

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Starting in 2015, microcephaly associated with Zika virus emerged as a public health emergency of international concern. Initial cases in the United States were travel-associated; however, there are increasing reports of local transmission in pockets of the country, and therefore public concerns may escalate.[1] Emergency physicians commonly perform point-of-care ultrasound (POCUS) on pregnant patients, a population of special concern. This paper describes ultrasound findings typical of Zika-related congenital malformations that may be incidental findings or detected when examining exposed or concerned patients during routine POCUS testing. These concerns should alert emergency physicians to assess for Zika-virus risk factors and provide urgent referral for confirmatory studies and counseling if indicated.

Fetal microcephaly and other congenital malformations are devastating consequences of Zika virus infection.[2] As the number of cases of Zika virus within the United States increases from either travel or local transmission, emergency physicians (EP) may encounter an increased population of pregnant females with a history of exposure to the virus either via mosquito bites, sexual transmission or possibly even transfusion-related. Pregnant patients may present to the emergency department (ED) either with perceived or real exposures to Zika virus infection. Point-of-care ultrasound (POCUS) performed either on these concerned patients or on any pregnant patient for routine indications may reveal incidental findings consistent with Zika-associated congenital abnormalities. As up to 80% of patients infected with Zika virus are asymptomatic, pregnant patients may be unaware they have contracted the virus.[3] Further, any pregnant female with a possible exposure to Zika virus may present with questions regarding the nature of possible complications as well as regarding follow-up care. Therefore, for purposes of counseling and referral, it is important that EPs be knowledgeable about ultrasound findings associated with congenital Zika virus infection as well as indications for urgent referral for confirmatory testing.

Ultrasound findings in fetuses with congenital Zika virus infection include microcephaly, intracranial calcifications, ventriculomegaly and arthrogyposis, as well as abnormalities of the corpus callosum, cerebrum/cerebellum and eyes (Images 1 and 2).[4,5] Fetal microcephaly is defined as a head circumference measurement either less than two standard deviations below the average head circumference or below the third percentile for sex and gestational age.[6] Microcephaly can be detectable as early as18-20 weeks gestational age. Diagnostic utility of US increases as gestational age increases. Even in the absence of microcephaly, the presence of intracranial calcifications prior to 22 weeks gestational age may predict its future development.[4] While presence of suspicious findings should prompt urgent referral, regardless of bedside US appearance,

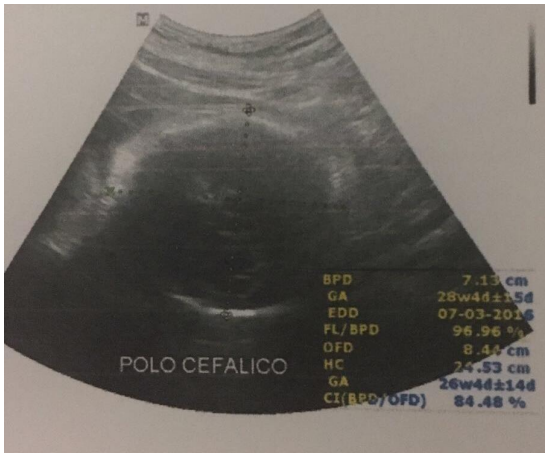

Image 1. Obstetric ultrasound image depicting fetal microcephaly in a mother with a history of Zika virus infection. The fetus is at approximately 34 weeks gestational age with a head circumference of 24.53 cm. The 50th percentile head circumference at 33 weeks and 6 days is around 30 cm (Image courtesy of Dr. Edson Silva, Rio de Janeiro, Brazil).[7]

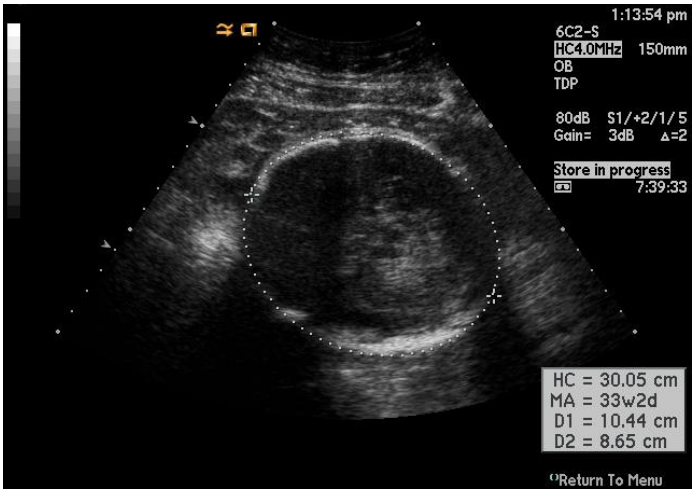

Image 2. Obstetric ultrasound image depicting normal fetal anatomy at a gestational age of approximately 33 weeks and 2 days, with head circumference of 30.05 cm (Image courtesy of http://www.learnobultrasound.com/datinggrowth.htm).

all women concerned about Zika virus infection should be referred to an obstetrician for further testing and formal US evaluation. Timing of exposure/presentation as well as serologic testing results will determine whether routine obstetric US or serial US are indicated.[5]

While the detection of specific point-of-care fetal ultrasound findings associated with Zika virus disease is beyond the scope of knowledge expected for EPs, any unusual appearance should prompt an assessment of the patient's risk factors for the infection. This is particularly critical as the vast majority of patients will be asymptomatic. Through knowledge of US findings indicative of Zika-virus related congenital malformations, EPs will be empowered to facilitate timely referrals for concerned or possibly exposed pregnant patients who may otherwise only present to the ED for routine POCUS testing.

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A Gift from Vacation: New Rash on His Foot

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A 32-year-old man sought care at the emergency department for evaluation of a rash on his foot, three weeks after returning from a beach vacation in the Caribbean. He reported that the rash had appeared one week earlier, was severely pruritic, and seemed to be expanding daily. He reported no systemic symptoms and had normal vital signs. Examination demonstrated two raised, erythematous, serpiginous lesions on the plantar aspect of his left foot (Imaage).

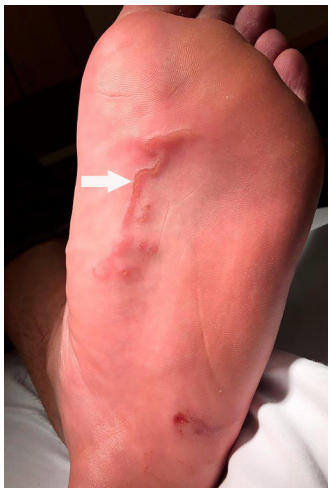

Image. Rash associated with cutaneous larva migran (arrow). Erythema on the heel is not part of the rash.

Cutaneous larva migrans (CLM) is an infection caused by several different types of hookworm. Infected animal hosts, usually cats and dogs, shed hookworm eggs in their feces. When CLM develops from animal sources it is often referred to as *hookworm-related CLM*.[1] The eggs hatch into larvae that can survive for several weeks under the right conditions – most commonly warm sand or soil in tropical or subtropical areas, including the Gulf Coast states. Larvae are most often transmitted to people walking barefoot through these areas, although hands and buttocks are also common sites of entry. The larvae release degradative enzymes to penetrate through the epidermis, which causes the characteristic rash several days to several weeks after exposure. The track may extend several millimeters per day as the larvae migrate through the skin.[2] Hookworm may cause systemic infections in animal hosts, but most species lack the collagenase necessary to penetrate the dermis in humans and are therefore confined to the skin.[3]

CLM is diagnosed clinically on the basis of the classic finding of a pruritic, serpiginous rash in the setting of recent travel to an endemic area. Although the infection is self-limited because the larvae die within five to six weeks, treatment is often required to address the severe pruritus. Oral antihelminthics (albendazole or ivermectin) are effective, possible therapeutic options.[1]

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Orbital Blowout Fracture From Nose Blowing

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A 38-year-old woman with a history of seasonal allergies presented to the emergency department with sudden onset of left periorbital swelling following nose blowing. There was no history of trauma, prior surgery, sinusitis or associated illness. On examination there was significant non-tender left periorbital swelling, with crepitus on palpation (Image 1). Extraocular movements, pupillary reflexes, fundoscopic examination and visual acuity were normal. Maxillofacial computed tomography (CT) showed subcutaneous emphysema (Image 2) and spontaneous left orbital floor blowout fracture with herniation of the orbital fat, and inferior rectus muscle and inferior oblique muscle into the left maxillary sinus (Image 3). Upon reevaluation, extraocular movements were normal. The patient was referred for oculoplastics evaluation.

Orbital emphysema is usually secondary to traumatic orbital fracture. In the absence of trauma, this is a rare condition. [1] Other mechanisms, including infection, pulmonary barotrauma, injury from compressed-air hoses, and complications from surgery and sneezing, have been reported to cause orbital emphysema.[2] Blow-out orbital fracture in absence of trauma is a rare condition as described in our patient. A few cases of orbital floor fracture following forceful nose blowing have been reported in the literature.[3,4] Blow-out orbital fractures most often involve the thinnest portions of the orbit, namely inferior (36.7%) and medial (31%) orbital wall.[2,5] A proposed mechanism for nontraumatic orbital fracture is that chronic maxillary sinusistis may weaken the orbital floor, making it more vulnerable to fracture by increased pressure following forceful nose blowing.[4] Patients should be instructed to avoid nose blowing, coughing or the Valsalva maneuver for at least two weeks after the injury.[6] There are many studies detailing the use of antibiotics in maxillofacial fractures, but very few on the use of antibiotics in isolated orbit fractures.[7] Patients with preexisting sinus disease may be at an increased risk for infection.[7] Prophylactic oral antibiotics (such as amoxicillin-clavulanate or azithromycin) to cover sinus pathogens are generally recommended for patients with orbital fracture into a sinus.[8]

In conclusion, emergency physicians should consider nontraumatic orbital fracture in patients with orbital emphysema with no history of trauma. CT is the recommended study of choice.

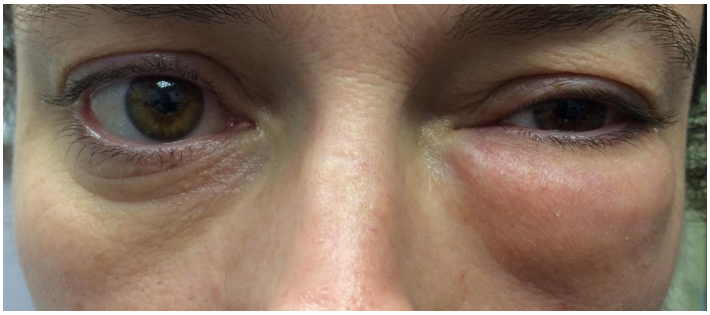

Image 1. Non-tender left periorbital swelling.

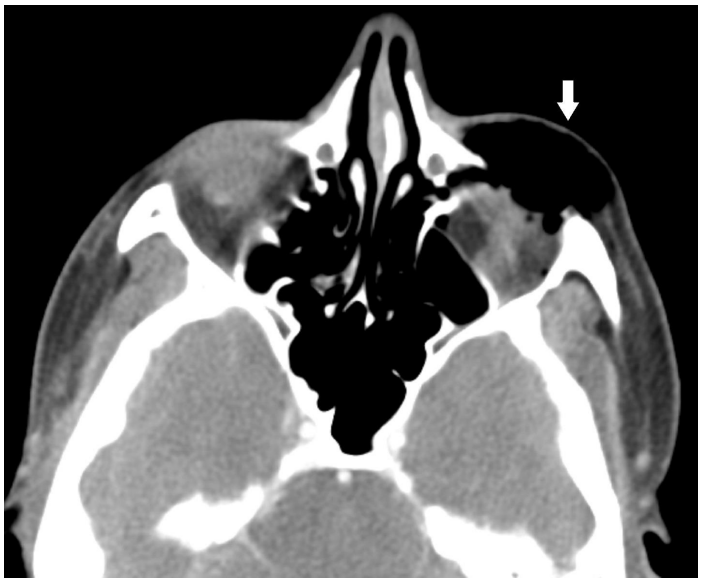

Image 2. Computed tomography shows subcutaneous emphysema around the left orbit (arrow).

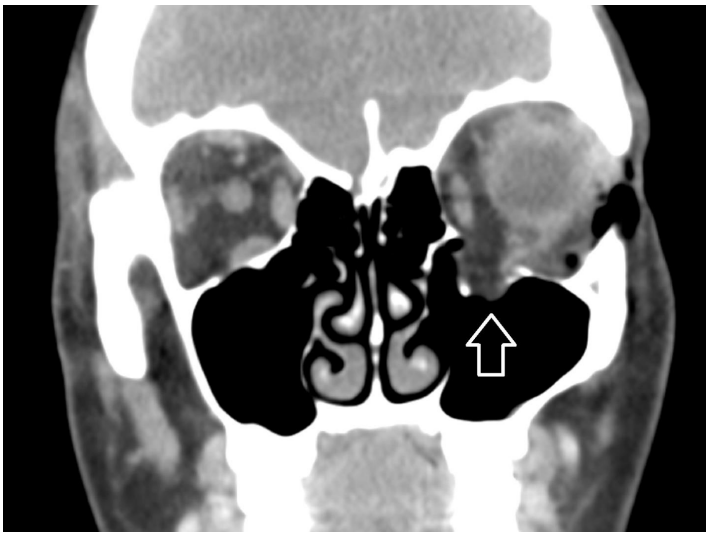

Image 3. Computed tomography shows left orbital floor blowout fracture with herniation of the orbital fat, inferior rectus muscle and inferior oblique muscle into the left maxillary sinus (arrow).

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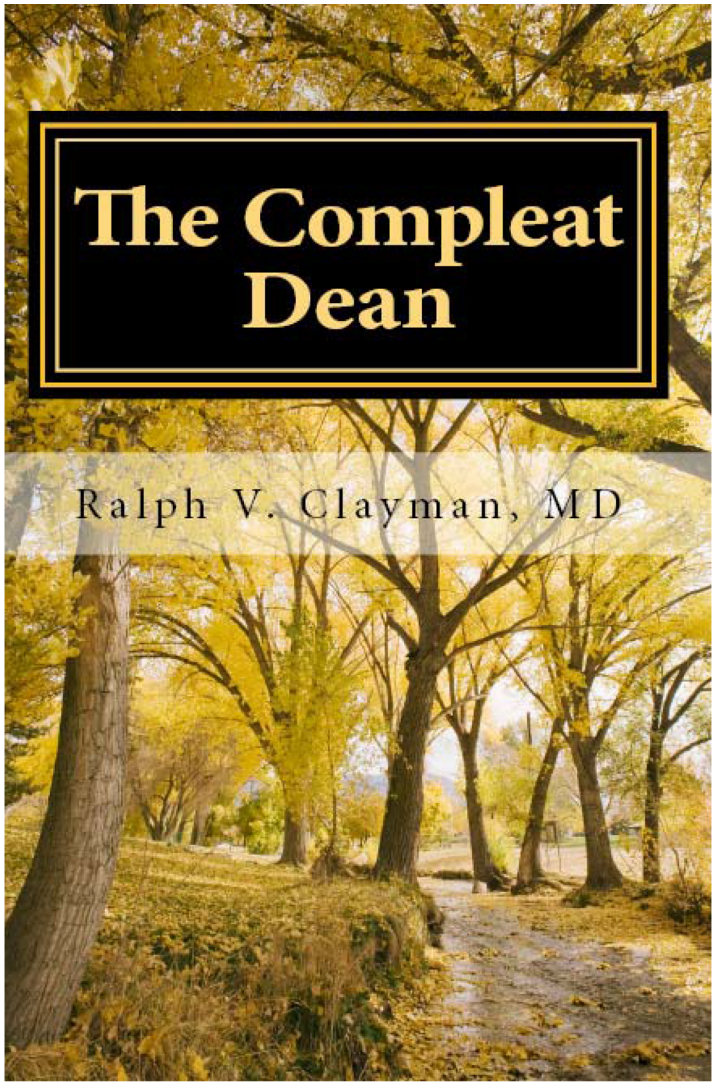

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